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Late Results in Benign Giant-Cell Tumor of Bone Obtained byRadiation Therapy[1]

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THE PRINCIPLES of therapy of giant-cell tumor of bone have been, from the beginning, subordinated to the variation in pathologic and histogenetic interpretation. In the early days, when the lesion was commonly regarded as belonging to the group of true bone sarcoma, radical surgery constituted the mode of attack.

In 1920, the Bone Tumor Registry of the American College of Surgeons, recognizing the benign character of most giant-cell-containing new growths of bone, placed them in a separate division under the name of "benign giant-cell tumor." Since then the trend of therapy has been toward conservatism. Inasmuch as metastases were not to be feared, attention was centered on how to apply the treatment locally in the most advantageous manner. Where formerly amputation or resection was advocated, curettage, swabbing of the bony cavity with escharotics, intracavitary radium, etc., were applied, the aim being to save the limb and if possible to restore its normal function. Later, when it was found that the curetted bony cavity was often slow in filling in and when a certain number of cases recurred or became secondarily infected, irradiation was tried.

Thus in its incipience, radiation therapy of giant-cell tumor of bone formed a supplement to surgical intervention, usually under most unfavorable circumstances. If one also considers the peculiar radiation reaction which is observed in this lesion, and which was not altogether understood at that time, there can be no doubt that the method was placed in a position of jeopardy even as to its merit in an auxiliary capacity. All too often the cases treated by such a combination had to be subjected in the end to amputation, a procedure which, in fact, signified complete failure of the local therapeutic effort.

Additional study of the radiation behavior of this tumor became necessary when it was unexpectedly noted that some of the patients who, after presumably unsuccessful treatment by curettage and irradiation, refused to submit to more radical surgery turned up later entirely recovered from their former trouble without further treatment. During the same time new concepts started to make a heavy inroad on the histogenetic interpretation of the lesion. The opinion was heard with increasing frequency that giant-cell tumors are merely the result of a foreign-body response or a repair process of the disturbed osseous structure and, since not all arise under the same circumstances, they may represent parts of a more com-

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plex agglomeration of tumors. It thus became apparent that radiation therapy in order to be of value must be carried out with a technic which would take into consideration special requirements.

These factors have given impetus to a long series of clinical investigations which now are apparently leading to the establishment of irradiation, in the form of roentgen therapy, as the primary method of treatment in all cases of benign giant-cell tumors and allied cystic conditions of bone.

HISTORICAL SURVEY

The medical literature is still rather meager in articles dealing with all phases, and especially the late results, of radiation therapy. Following a few individual case reports Herendeen (10) in 1924 first published early satisfactory results in a larger group of cases, 16 of which were treated by irradiation alone. One of his patients had been well for five and a half years at the time of the publication and on this basis Herendeen predicted that in most of the cases a complete cure and restoration of function could be anticipated. In subsequent publications (11, 13) these good results were confirmed.

In 1928, Lacharité (15) reported a series of 14 cases, 12 of which were tumors of the epulis type of the maxilla and mandible. They were treated by radon molds or seeds or by roentgen therapy at the Radium Institute of Paris, all with satisfactory results. Later, Pfahler and Parry (21) added a group of 26 cases, 17 patients being traceable and under observation as "well." In this series, 14 patients were operated upon before irradiation was given and 12 were treated by radiation therapy alone. Although it was found impossible to give the final results satisfactorily in any tabular form, the conclusion was drawn that irradiation was definitely superior to surgery and that crushing of the bone or curettage was of no advantage, but rather a disadvantage. One of the cases included in this group was irradiated by Pfahler as early as March 1906, and it apparently represents the first case of giant-cell tumor treated by radiation therapy. The patient was well twenty-five years later.

In 1936, Peirce and Lampe (20) reported on a group of 27 cases treated since 1923. Three of these were treated by surgery alone, 12 by a combination of surgery and radiation therapy, and 12 by irradiation alone. Though lack of uniformity of the cases defeated attempts at absolutely rigid comparison, nevertheless it was thought that roentgen irradiation offered the most to the patient, except when, for cosmetic or functional reasons, better results could be obtained with the addition of surgical intervention.

Coley and Higinbotham (4), who in 1938 published the results in a group of 103 cases, have taken a compromising attitude, giving surgery the preference in all accessible giant-cell tumors and reserving radiation for the inaccessible and extremely advanced tumors. Of 30 cases treated by surgery alone, 23 were cured locally, whereas of 32 patients treated by a combination of surgery and radiation therapy, only 15, and of 41 treated by irradiation alone, 26 got well. An additional 21 cases were eliminated from the final tabulation because of various extraneous factors.

There are, of course, a number of articles from such eminent investigators as Bloodgood (1), Geschickter and Copeland (9), Linde (17), Meyerding (18), Freund and Meffert (8), and others, which deal *in extenso* with all phases of the surgical treatment of giant-cell tumor of bone. In some of these, the radiotherapeutic aspect is also considered, but the discussion not infrequently is based on speculative criteria and a rather limited experience with a small group of cases, as a rule representing surgical failures or very advanced tumors, in which irradiation was applied in a secondary rôle. For example, in Bloodgood's series of 77 cases, Linde's collection of 14 cases, and Meyerding's group of 61 cases, none was treated by irradiation alone; of Geschickter and Copeland's material of 214 cases since 1896, roentgen

therapy constituted the sole method of approach in only 5, and of the 15 cases tabulated by Freund and Meffert, in only one. Even the articles which base their study on the material of the Bone Tumor Registry, such as the analyses of Kolodny (14), Simmons (22), Codman (3), and others, fail to give an entirely clear evaluation of the merit of radiation therapy, although its place in the treatment armamentarium is admitted and of late some concession is made as to its possible future rôle as a primary method of approach.

On the other hand, Doub, Hartman, and Mitchell (5) found that in a group of 22 cases of the Bone Tumor Registry treated by radiation therapy alone or in association with surgery, 3 later became malignant. Since the same thing happened in one of their own cases, they raised the question whether small, frequently repeated doses of roentgen therapy may not have had a stimulative effect on the tumor cells, leading, especially when the application was made in conjunction with traumatizing surgical procedures, to malignant transformation.

Because of this rather doubtful position of irradiation in the treatment of giant-cell tumors of bone, it occurred to the present authors that the publication of the late results in an additional group of cases might contribute, in whatever small measure, to the further elucidation of the relative value of the method. At the same time, the consideration of certain special phases may help to create more rigid criteria of technical procedure and clinical interpretation.

MATERIAL

The study is based on a total of 33 cases treated at Harper Hospital, Detroit, from 1923 to 1935, inclusive (see Table I). In the entire group, death from sarcomatous degeneration occurred once (Case 10, Fig. 15). Two patients died from incidental causes, though there was a satisfactory local result, and one case was untraced.

The method of treatment from 1923 to 1929 consisted, in most instances, of curettage and postoperative irradiation. Of 13 cases so handled, amputation had to be performed later in 3 cases and in a fourth case there occurred three years after the curettage a sarcomatous degeneration, with death. Thus in this group 30 per cent of the cases represented a failure eventually of the local therapeutic effort. Since 1930, radiation therapy has constituted the method of choice. Of a total of 20 cases, curettage was done in only 2 and biopsy was taken in another 3; 15 cases were subjected to no surgical procedure. In this group, a satisfactory local result was obtained in every instance, so that the patients completely regained the normal function of the affected bone. Two patients died later from incidental causes, one seven years after the treatment and the other about one year later. The one untraced case dates from 1929, but it is probable that this patient is well, both locally and generally, since at the time of his last visit he was on the road to recovery.

ADVANTAGES OF RADIATION THERAPY OVER SURGERY

In analyzing the above results as to the late anatomic appearance and functional restoration of the affected bone, the superiority of radiation therapy over surgical procedures at once becomes apparent. There are at least three reasons for this:

(1) Infection is not an altogether rare surgical complication even under most scrupulously observed aseptic conditions, whereas with radiation therapy it does not exist. As already stated, the more radical operations of the pre-registry era have gradually given place to curettage in the surgical treatment of most benign giant-cell tumors. But to be efficacious, this procedure, too, had to be carried out with thoroughness, the aim being to remove the tumor as completely as possible. To forestall recurrence, then, the curetted cavity was swabbed with pure carbolic acid and alcohol or with zinc chloride. As a rule, the wound was closed, but if the cavity was very large and there was a danger of

TABLE I: SUMMARY OF CASES

Year	No.	Sex and Age	Site of Lesion	Histologic Diagnosis	Treatment	Local Result	End-Result	Illustration
1923	1	F 16	Left mandible	Epulis	I: 6/15 DRT 100% SUD; 8/9 curettement. II: 8/13 Intracavitary Ra 1,350 mg.-hr.; 8/17 DRT 90% SUD	Good	W.	
	2	F 20	Left lower femur	Giant-cell tumor	1/15/22 curettement; 6/25/23 curettement; I: 7/16 DRT 90% SUD. II: 9/24 70% SUD; amputation later	Amput.	W.	
1924	3	F 25	Right maxilla	Epulis	I: 7/10 DRT 110% SUD; 7/14 curettement, intracavitary Ra 600 mg.-hr.	Good	W.	
	4	M 29	Left lower femur	Giant-cell tumor	4/13 Biopsy. I: 4/18 DRT 110% SUD. II: 6/2 100%. III: 7/15 100%; infection of cavity; 5/14/25 amputation	Amput.	W.	
1925	5	F 48	Left thumb	Giant-cell tumor	12/4 Curettement. I: 1/20/25 DRT 100% SUD. II: 9/2 110%	Good	W.	Fig. 1
	6	F 5	Left antrum	Epulis	5/20 Curettement. I: 6/8 DRT 110% SUD	Good	W.	
1926	7	M 32	Right lower radius	Giant-cell tumor	6/14 Curettement. I: 6/24 DRT 110% SUD. II: 10/13 70%; 10/17/27 2nd curettement. III: 10/28 DRT 100% SUD	Good	W.	Fig. 2
	8	F 33	Left 3d metacarpal	Giant-cell tumor	1/15 Curettement. I: 2/3 DRT 110% SUD; 7/19 amputation of metacarpal and 3d finger. II: 7/19 Intracavitary Ra 1200 mg.-hr.; infection; in summer of 1927 amputation of hand	Amput.	W.	
	9	F 32	Right lower ulna	None	I: 1/5 DRT 80% SUD. II: 2/17 60% SUD. III: 4/22 30% SUD	Good	W.	
1927	10	M 24	Right upper tibia	Giant-cell tumor	In 1925, 3 curettements. I: 9/6/26 DRT 90% SUD. II: 11/5 90%. III: 12/24 90%. IV: 2/11/28 50%. V: 4/2 50%. In 1932 sarcomatous degeneration. Died Jan. 1933	Sarcomatous degeneration	D.	Fig. 15
	11	M 29	Right upper radius	None	I: 3/8 DRT 90% SUD. II: 4/18 90%. III: 6/14 80%. IV: 8/7 70%. V: 10/4 60%	Untraced		
1929	12	M 7	Left upper humerus	Giant-cell tumor	6/17 Curettement. I: 6/17 DRT 60% SUD. II: 7/27 40%. III: 10/10 30%	Good	W.	Fig. 3
	13	M 27	Right upper tibia	Giant-cell tumor	7/26 Curettement. I: 8/20 DRT 60% SUD. II: 10/15 50%. III: 12/16 40%. IV: 2/15/30 40%. V: 4/15 30%	Good	W.	Fig. 4
	14	M 19	Greater trochanter right femur	Giant-cell tumor	6/13 Curettement. I: 8/27 DRT 100% SUD. II: 10/20 80%. III: 12/15 80%	Good	W.	Fig. 5

1930	15	F 17	Left mid humerus	None	I: 7/21 DRT 80% SUD. II: 9/19 90%. III: 11/29 60%. IV: 1/29/31 50%	Good	W.	Fig. 6
	16	M 42	Left lower femur	None	I: 7/29 DRT 100% SUD. II: 9/6 90%. III: 9/28 90%. Incidental death 1937	Good	D.	
	17	M 27	Lower cervical and upper dorsal spine	Giant-cell tumor	12/23/29 Biopsy. I: 4/7/30 colloidal lead and DRT, 110% SUD. II: 6/3 100%. III: 8/6 70%. IV: 10/28 60%	Good	W.	Fig. 7
	18	F 33	Left antrum	None	I: 3/30 Intracavitary Ra 850 mg.-hr. and DRT 130% SUD	Good	W.	
1931	19	F 45	Left antrum	Epulis	3/10 Biopsy. I: 3/19 contact Ra 500 mg.-hr. II: Contact Ra 300 mg.-hr.	Good	W.	Fig. 8
	20	F 75	Right index finger	None	I: 6/1 DRT 70% SUD. II: 7/13 70%. III: 9/14 70%. Incidental death 1932	Impr.	D.	
	21	F 7	Left upper humerus	None	I: 8/3 DRT 80% SUD. II: 8/30/32 30% SUD. III: 10/26/32 30% SUD. IV: 12/29/32 30% SUD	Good	W.	Fig. 9
1932	22	M 22	Right lower tibia		I: 9/7 DRT 60% SUD. II: 10/19 60%. III: 12/7 50%. IV: 2/8/33 40%. V: 5/3 40%. VI: 8/29 40%	Good	W.	
	23	M 52	Left mandible	None	I: 10/22 Ra puncture 300 mc.-hr.	Good	W.	
	24	F 26	Lower incisors	Epulis	6/30 Curettement. In fall, root canal filling of incisors. In 1934 apicalectomy	Good	W.	
1933	25	F 11	Mental region of mandible	Epulis	7/27 Biopsy. I: 8/17 DRT 70% SUD. II: 10/5 60%. III: 12/6 40%. IV: 2/6/34 30%. V: 4/3 30%. VI: 6/13 20%. VII: 12/1 20%	Good	W.	
	26	F 6	Right upper femur	None	I: 12/6 DRT 60% SUD. II: 2/7 50%. III: 5/21 40%	Good	W.	Fig. 10
1934	27	F 1	Right upper femur	None	I: 3/22 DRT 60% SUD. II: 5/9 50%. III: 7/9 30%	Good	W.	Fig. 11
	28	F 46	Left index finger	None	I: 2/5 DRT 70% SUD. II: 3/21 60%. III: 5/27 50%. IV: 12/20 30%	Good	W.	
	29	F 57	Right index finger	None	I: 9/6 DRT 70% SUD. II: 10/11 70%	Good	W.	
	30	F 33	Left mid finger	None	I: 3/17 DRT 70% SUD; incomplete treatment	Impr.	W.	
1935	31	M 22	Right 1st metacarpal	None	From 12/5/35 to 9/11/38, 12 series of DRT with doses decreasing from 70 to 10% SUD	Good	W.	Fig. 12
	32	F 22	Left lower femur	None	From 3/2/35 to 1/8/37, 9 series of DRT with doses decreasing from 70 to 10% SUD	Good	W.	Fig. 13
	33	M 35	Right lower femur	None	From Dec. 1935 to 11/11/37, 10 series of DRT with doses decreasing from 70 to 10% SUD	Good	W.	Fig. 14

Abbreviations: DRT = Deep roentgen therapy with 200 kv., 1–1.5 mm. Cu and 1.0 mm. Al. Ra = Radium therapy, usually intracavitary with 1.0 mm. brass and 1.0 mm. hard rubber filter. SUD = Skin unit dose, 100% representing for 200 kv. (1 mm. Cu) 900 r on skin or tumor (525 r in air) given in one séance over 20 × 20 cm. field at 20 r/min. intensity. W. = Well. D. = Died.

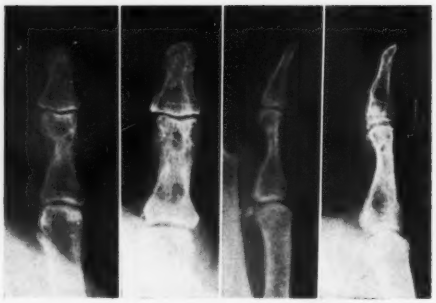

Fig. 1 Case 5: Giant-cell tumor of thumb before and fifteen years after curettage and roentgen therapy. Note soft tissue swelling.

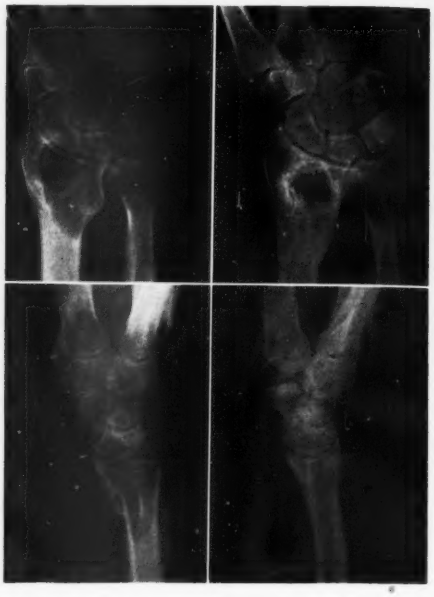

Fig. 2. Case 7: Giant-cell tumor of lower end of radius before and fourteen years after two curettements and roentgen therapy. Some bone defects still present.

hemorrhage, the curetted area was packed with gauze, which was later removed, or with some material such as bismuth paste (Fig. 15), which was left *in situ* indefinitely. In still other instances, when the application of chemical or thermic escharotics was suspected not to have acted deeply enough to destroy all of the possible remaining tumor cells, radium tubes were placed intracavitarily for a certain length of time.

It has already been demonstrated elsewhere (6) that such measures not infrequently led to some infection of the curetted cavity and surrounding healthy bone, eventually necessitating the performance of a resection or amputation. Since then, statistical data continue to show the same relatively high incidence of this complication so that Ewing (7) not so very long ago was prompted to say: "I have seen so many bad results from recurrence, malignant transformation, infection of all types, acute and chronic, resulting in amputation, that I have no longer any room to file these cases in our museum."

Infection with subsequent amputation occurred in 2 out of 13 cases of our earlier series, despite the addition of radiation therapy. In a third case, amputation had to be performed several years later because of the recurrence of the giant-cell tumor.

The high vascularity of the tissues of the average giant-cell tumor and the free exposure of the bone marrow are factors which predispose to secondary infection even in a simple biopsy. For this reason, and since roentgen study of the affected bone usually permits a rather accurate diagnosis especially as far as a differentiation from osteogenic sarcoma is concerned, not a few of the investigators prefer to dispense with the microscopic examination, except in doubtful instances.

In 15 cases of our series so handled, good local end-results were obtained without inconvenience as to the lack of histologic study, and since in none of the cases did metastases develop later, it was assumed that the clinical diagnosis was correct.

(2) Another disadvantage of the surgical treatment of the benign giant-cell tumor lies in the fact that a thorough curettage when associated with chemical or thermic escharotics leads to a complete destruction of the trabeculations of the bone within the affected area. As a result of this, the process of bone regeneration is greatly prolonged and the filling in of the tumor cavity not infrequently re-

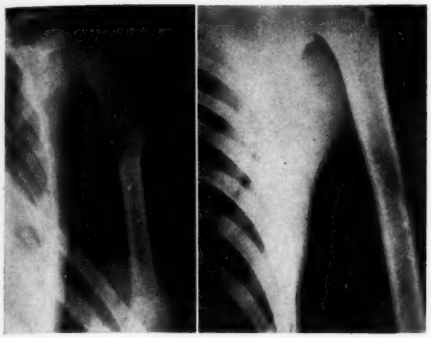

Fig. 3. Case 12: Giant-cell tumor of upper third of left humerus. Left: Status after curettage and before roentgen therapy. Right: Eleven years later.

mains incomplete. An analysis of some of our earliest cases (Figs. 2 and 4) shows, for example, that as late as fourteen years after treatment, osseous defects may still be demonstrable on the roentgen films, although the normal function of the limb may have been regained. In the cases treated by irradiation alone, the reossification occurs somewhat more rapidly and in the end a more compact restoration of the bony framework is observed.

Brunschwig (2), on the basis of a study of 9 cases observed periodically for a longer interval, expresses the opinion that this difference may be attributed to the variation in the healing process. In the curetted cases, a large blood clot is formed following the operation. Thus the subsequent changes are those of an organizing hematoma through which calcification follows. In the cases treated by roentgen therapy, the reossification is the result of metaplasia in the tumor, an effect of the irradiation.

(3) A third disadvantage of treatment by curettage and escharotics is the fact that repeated intervention in case of recurrence may act as trauma, leading to possible malignant transformation. It is significant in this respect that the only case in our series which has shown sarcomatous degeneration (Case 10) has had 3 curettements, bismuth paste was placed permanently in the non-filling bony cavity

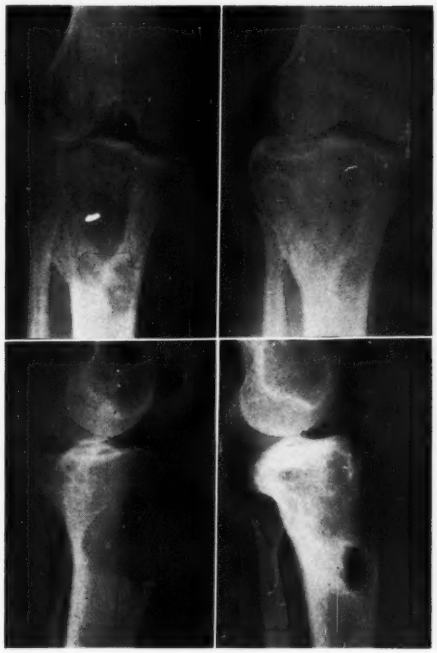

Fig. 4. Case 13: Giant-cell tumor of upper end of right tibia, before and ten years after curettage and roentgen therapy. Some bone defects still present.

(Fig. 15), and finally the patient himself suffered an accidental fracture through the tumor area, a few months before the malignant transformation occurred.

Similar rare cases of sarcomatous degeneration due to repeated surgical trauma have been described by Stone and Ewing (23), Peirce (19), Herendeen (12), and others.

INDICATIONS FOR RADIATION THERAPY

Apart from its advantages over surgery enumerated above, radiation therapy is indicated as the primary method of approach in the giant-cell tumor and allied cystic conditions of bone because of its simplicity and complete innocuousness to the surrounding normal structures and because, in case of occasional failure, surgical intervention can always be substituted later. Since these lesions are with few exceptions benign, no time is wasted by first employing irradiation, which may

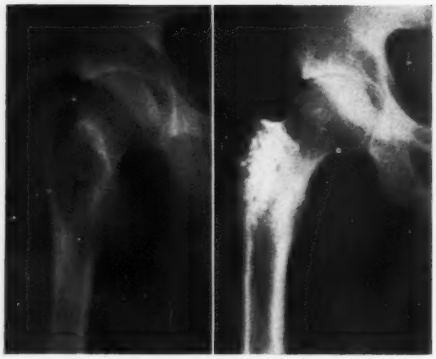

Fig. 5. Case 14: Giant-cell tumor of greater trochanter of right femur before and ten years after curettage and roentgen therapy.

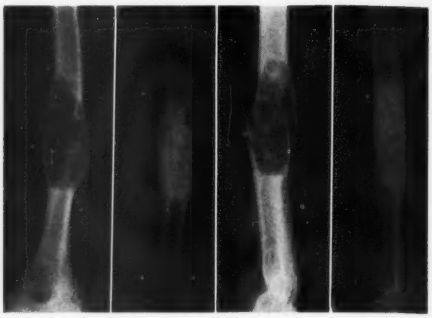

Fig. 6. Case 15: Giant-cell tumor with pathologic fracture of mid-shaft of left humerus before and ten years after roentgen therapy. No biopsy.

lead to complete restoration of the function of the affected bone, and reserving the operative procedures which so often terminate in the sacrifice of the limb to the very last. Furthermore, in those rare instances in which sarcomatous degeneration has already occurred, or in which the possibility of a primary sarcoma of the bone cannot be excluded on the basis of the roentgen examination alone, the malignant process is usually of the osteolytic or telangiectatic type so that here, too, radiation therapy is to be preferred to surgery. It remains a fact that in none of our 15 cases diagnosed without the aid of biopsy was there evidence of malignancy later, and the local end-results obtained by the irradiation remained very satisfactory.

APPLICATION OF RADIATION THERAPY

In applying radiation therapy to the giant-cell tumor of bone, two factors must be taken into consideration: (1) the adaptation of the technic to the rather pronounced radiosensitivity of the tumor tissues and the slow reossification of the destroyed area, and (2) the special management of the patient during this period.

Technic of Irradiation: Based on the acknowledgment that giant-cell tumor represents a benign growth, irradiation is carried out with a local technic, attention being paid only to the site of the primary lesion. During the course of time some changes have been made, however, especially in the dosage and duration of the treatment. Curiously enough, these changes coincided with the variation in the histogenetic interpretation of the tumor, although they were motivated by clinical experience rather than the consideration of such factors.

The problem of histogenesis of the giant-cell tumor, as Kolodny (14) states, follows an old trodden path of discussion. On the one hand are those who consider the giant-cell tumor a true neoplasm of unknown etiology, but with the potentiality of every progressive or regressive change that usually occurs in blastomata. On the other are the opponents of such an interpretation, who advance the view that giant-cell tumor is merely a product of inflammation and repair and that the term "tumor" as used here signifies only progressive expansion. In a broader sense, one may include the regressive changes leading to cyst formation in osteitis fibrosa or some other localized osseous change in the same category.

However this may be, the large doses which were given in the beginning, on the basis that the condition is a true neoplasm which must be eradicated as thoroughly and in as short a time as possible, have gradually given place to the application of smaller doses and prolongation of the treatment over a considerably greater period. Obviously such an approach in-

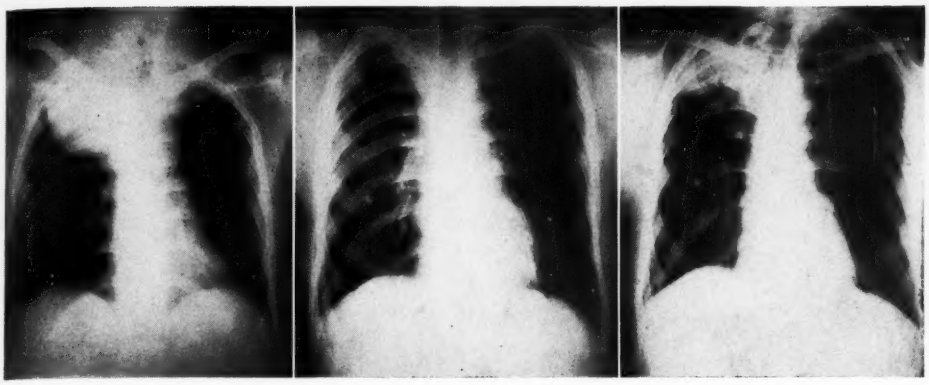

Fig. 7. Case 17: Giant-cell tumor of lower cervical and upper dorsal vertebrae with transverse block of spinal cord and complete paralysis from the waist down. Left: Status after laminectomy and biopsy. Middle: Same one year after roentgen therapy. Right: Same ten years after roentgen therapy. Slight fibrosis of lung. Complete return of normal function.

directly supports the inflammation or repair process theory of the benign giant-cell tumor and related cystic conditions of the bone.

(*a*) *Dosage:* Herendeen (10), who was one of the first to advocate irradiation with large doses over a short interval, described very vividly the phenomena that followed such a procedure. Within one or two months after the first series of treatments, the tumors rapidly increased in size, the skin became reddened and edematous, the bony cortex expanded and thinned, and the osseous trabeculations within the affected area were so washed out that hardly any bony detail was visible. The general impression was that the irradiation aggravated the condition rather than improved it, and it was only after many months, following additional radiation therapy, that a change for the better was noted, eventually leading to ossification of the involved area with restoration of normal function. This post-radiation reaction constituted at that time, as may be surmised, a continuous source of misinterpretation. Radical operation was recommended, and often carried out, when this was not necessary, simply because radiation therapy was thought to be a failure. This has happened also in some cases of our series. Two patients (Cases 9 and 18), however, refused to submit to surgery and to our great surprise were found later to have completely recovered, without further treatment than the initial radiation therapy.

After several years' clinical experience, Herendeen (12) resorted to smaller doses, feeling that severe reactions were unnecessary. In his last publication, in 1931 (13) he advocates a dose seldom approaching an erythema, the factors being: 140 kv., 4 ma., 4 mm. Al, 10–12 in. skin-target distance, 12–15 min. exposure, a series consisting of 3 treatments, from three days to one week apart, an average of 8 to 10 treatments being given.

Pfahler and Parry (21) use deep roentgen therapy with 200 kv. and 0.5 mm. Cu forefilter, 25 per cent skin erythema doses being given at a distance of 50 cm., through one, two, three, or four fields of entry, as a rule three times a week (or in case of doubtful malignancy daily) until the tumor area has received a 100 per cent skin erythema dose according to the "saturation technic of Pfahler." Six weeks or two months after the completion of the first series, the case is re-studied and treated accordingly. Degenerative effects from the irradiation are scrupulously avoided.

A change from low-voltage (120–140 kv. peak, 0.25 mm. Cu) to high-voltage (200 kv. peak, 1 mm. Cu) technic was made in the cases reported by Peirce (19). The

doses are conservative—larger single (up to 60 per cent SUD) or smaller multiple exposures during the first weeks or months, gradually shading off as the tumor decreases and bone production increases.

In our series of cases, the quality of the roentgen rays was that obtained with 200 kv. equiv. (1 mm. Cu forefilter) from the beginning. The initial dose first was in the neighborhood of 80–90 per cent SUD,[2] but since 1932 it exceeds 70 per cent SUD only in case of doubtful malignancy. At subsequent series, the dose is gradually reduced at the rate of about 10 per cent per series until the irradiation is completed. The full series is given in a single séance, as many fields being used for the purpose of cross-firing as is necessary to get a fairly uniform distribution over the tumor area. The series is repeated within two months, and at gradually increasing intervals thereafter.

During the past few years, roentgen rays obtained with 500 kv. equiv. (7 mm. Cu) were used more frequently, for at increasing voltages a larger physical dose can be administered to the tumor with less damage to the normal structures. Inasmuch as the aim is to obtain a good local result, the skin can be saved in this manner to greater advantage.

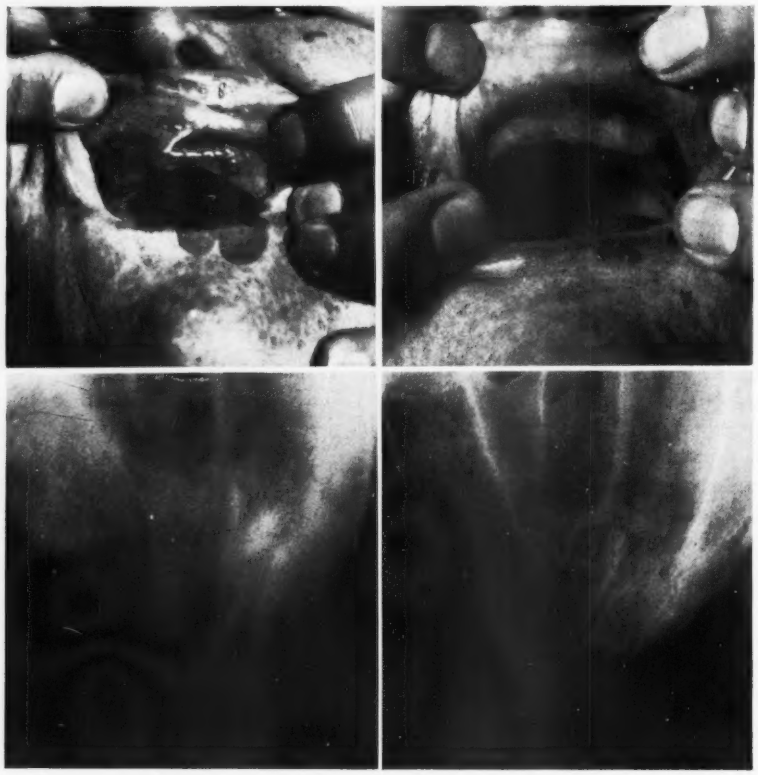

Fig. 8. Case 19: Epulis of left maxilla before and nine years after biopsy and contact radium therapy.

(*b*) *Duration of Treatment and Bone Regeneration:* The irradiation is continued

[2] One hundred per cent SUD represents 900 r (525 r in air) given in one séance on a field 20 × 20 cm. with an intensity of 20 r/min., if the quality of the roentgen rays is that obtained with 200 kv. equiv. (1 mm. Cu); and 1100 r (800 r in air) if the quality is that obtained with 500 kv. equiv. (7 mm. Cu).

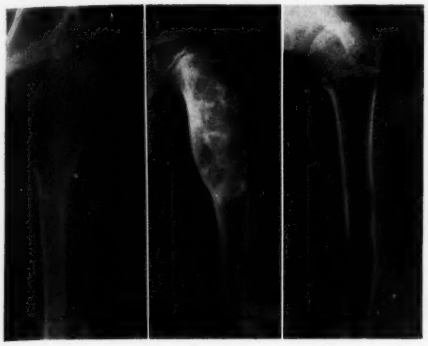

Fig. 9. Case 21: Giant-cell tumor of left upper humerus with pathologic fracture, before, two years, and nine years after roentgen therapy. No biopsy. The site of lesion moved to the middle of the shaft due to osteogenesis.

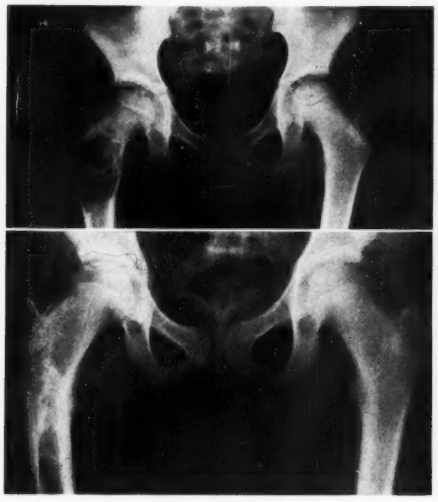

Fig. 10. Case 26: Giant-cell tumor of right upper femur with pathologic fracture, before and seven years after roentgen therapy. No biopsy.

as a rule until a fairly good reossification of the bone is obtained. On an average a period of two years is required for this purpose. It is not uncommon to see a patient receive as many as six to eight series of treatments before the final result is accomplished. If the dose of the initial series represented 70 per cent SUD, 60 per cent SUD will constitute the dose of the second series two months later, 50 per cent SUD of the third series three months later, and so on until the irradiation is completed.

It is interesting that the ossifying capacity of the bones varies, among other things, with the location and especially with age. It is our impression that in the adult bone regeneration occurs most rapidly in the flat bones. Next in line come the long bones, with the shafts showing a slightly higher and the epiphyseal ends a quite low recuperative capacity. The short bones display the longest period of regeneration of all. We have observed, especially in the phalanges of digits and the metacarpals, a continuation of the ossifying process for five years or longer (Fig. 12, Case 31), during which time, apart from the swelling, no other clinical symptom was present. The knowledge of this is important, since one may be tempted to resort to more radical surgical procedures, as, for example, resection or amputation, when in fact no such intervention is necessary (Fig. 1).

The influence of age on the regenerative capacity of bone is brought out most strikingly in children. Here one repeatedly sees such remarkable restoration of the normal osseous architecture that in later life the site of the largest tumors becomes barely visible on the roentgenograms (Figs. 9 and 11).

(c) *Procedure in Children:* With accumulation of a larger number of cases, it becomes increasingly apparent that the giant-cell tumors of children are the product of inflammation and repair. Their simultaneous occurrence with cystic conditions and frequent extent over a large portion of the bone without corresponding increase in the cortical expansion constitute further points for the support of such a view. But most important of all is the fact that they can be made to disappear following the administration of considerably smaller doses of roentgen rays than would be required if they were true blastomata.

Because of this and on account of the remarkable regenerative property of the bone, the procedure in children has been

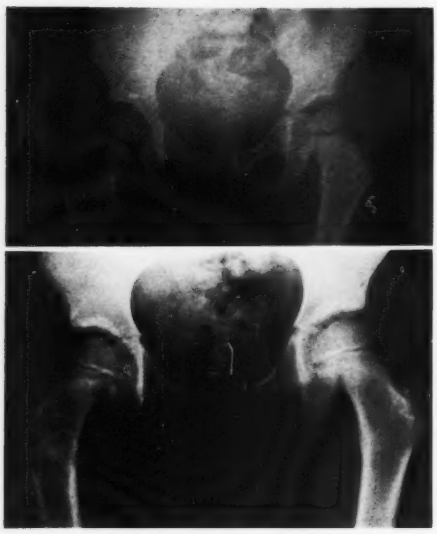

Fig. 11. Case 27: Osteitis fibrosa cystica of right upper femur before and six and a half years after roentgen therapy. No biopsy.

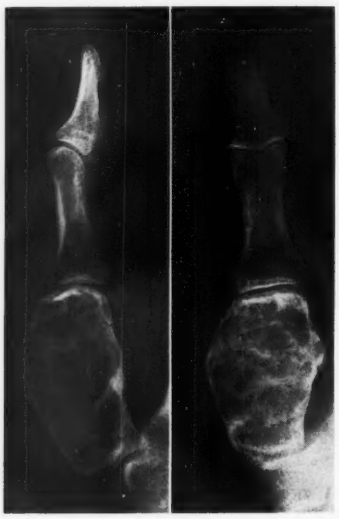

Fig. 12. Case 31: Giant-cell tumor of right first metacarpal with pathologic fracture before and five years after roentgen therapy. No biopsy. The reossification is not yet complete.

somewhat modified from that for adults in similar situations. The irradiation is carried out with the smallest doses possible, the intervals between the series being increased and the total number of series reduced according to circumstances. During irradiation the surrounding normal structures, especially the neighboring epiphyseal lines, are heavily protected, the fields being made as small as is compatible with the uniform distribution of the rays. To avoid interference with the remaining architectural frame of the affected bone, a biopsy is taken only in exceptional instances. All lesions, whether they be classified as giant-cell tumors, bone cysts, or localized osteitis fibrosa, are treated more or less alike.

In conformity with these principles, the initial dose is about 50 per cent SUD. The reduction for the subsequent series is made at the rate of 10 per cent, and the intervals between series are increased by several months as compared to adults. The total number of treatments is limited to as few as possible. Cases 12, 21, 26, and 27 (Figs. 3, 9, 10, and 11) represent examples of the good results obtained with such a technic, although in some of them the dosage and total amount of radiation were somewhat larger than would be used today.

LeWald (16) in 1930 reported 4 cases of giant-cell tumor and osteitis fibrosa cystica occurring in children seven to eleven years of age, in which satisfactory improvement was found three to three and a half years after treatment with moderate doses of roentgen rays obtained with only 120 kv., 4 mm. Al.

(2) *General Management:* Since giant-cell tumor and allied cystic conditions of the bone as a rule have an asymptomatic clinical course, the general management of the patient concerns itself chiefly with protection of the weakened structure of the affected bone during the process of healing. If a pathologic fracture has already occurred, the correction of the alignment of the bony fragments is necessary.

During the earlier days, and especially when extensive curettage with creation of large hollow cavities preceded radiation therapy, the common practice was to apply a supporting cast of plaster of paris, which

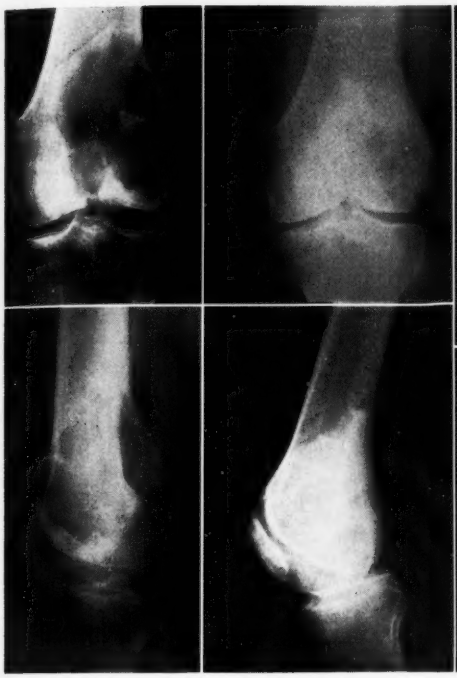

Fig. 13. Case 32: Giant-cell tumor of left lower femur with pathologic fracture into knee joint before and five years after roentgen therapy. No biopsy.

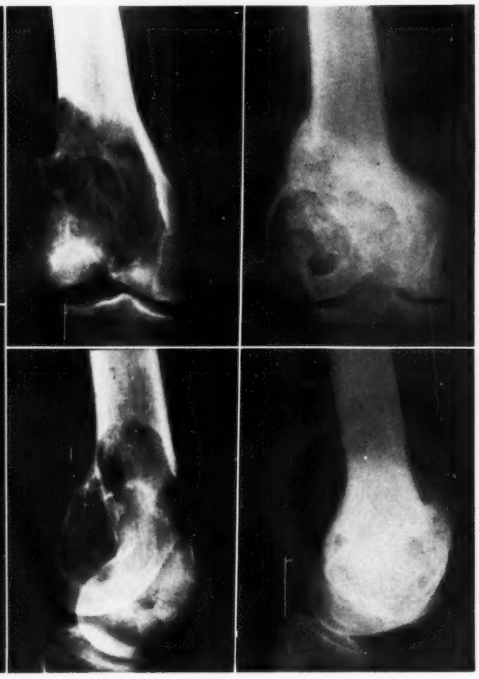

Fig. 14. Case 33: Giant-cell tumor of right lower femur with two consecutive pathologic fractures before and five years after roentgen therapy No biopsy.

usually included as much of the normal bone as was compatible with the idea of complete immobilization. If the lesion involved the long bones of the lower extremities, the distant portion of the limb was also placed in the cast, and if the process was located along the weight-bearing spinal column or in the bones of the pelvic girdle, a large plaster jacket covering the entire trunk was used.

It was soon found, however, that if the immobilization is carried out over a long period, the osseous demineralization incident to disuse assumes such proportions that it seriously hampers bone regeneration within the tumor-destroyed area. For this reason, during the past ten years we have dispensed with the application of plaster bandages. The patient is permitted to have free use of the affected limb, but is cautioned to be exceedingly careful because of the possibility of a pathologic fracture. If the giant-cell tumor is in the line of weight-bearing function, he is allowed to move about with the aid of a cane, crutches, or a walking Thomas splint. In rare instances, he is put to bed for several months, without more immobilization than the application of a few sandbags around the affected part. By following such a procedure, we have observed the occurrence of subsequent fracture in only one case (Case 33). This patient, an habitual alcoholic, suffered two successive fractures, each time due to carelessness. The final result, however, even here remained satisfactory (Fig. 14).

If pathologic fracture complicates the picture of the giant-cell tumor and allied conditions of the bone from the beginning, or at any time during the course of irradiation, a supporting plaster cast is applied for a limited period. Reduction of such a fracture, as a rule, does not offer great

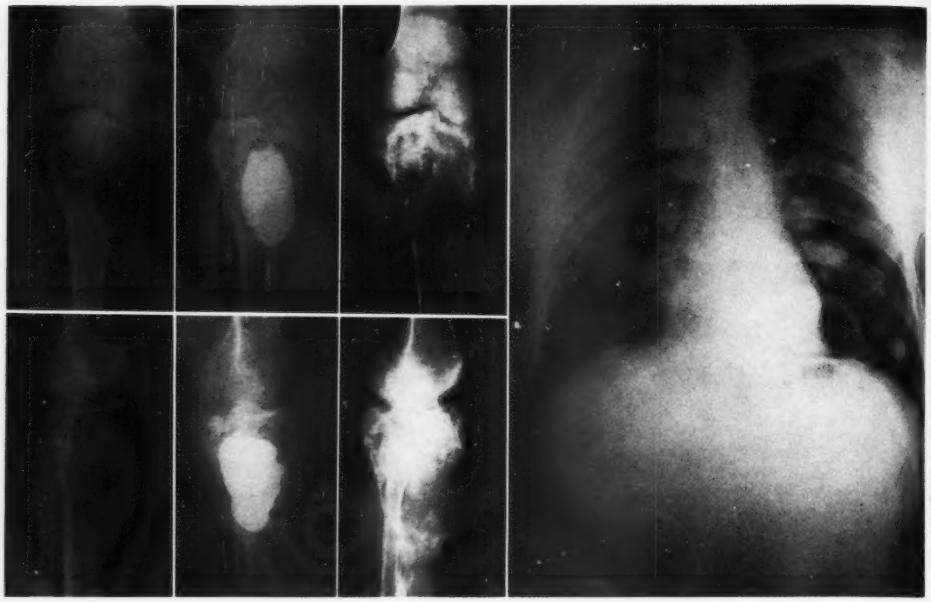

Fig. 15. Case 10: Giant-cell tumor of right upper tibia. In 1925, three curettements and filling of bony cavity with bismuth paste. From 1926–1928 roentgen therapy. In 1932 accident with fracture at site of tumor, followed by sarcomatous degeneration. Patient died in 1933.

difficulty, partly because of the ease with which the fragments can be approximated and partly because of the relative lack of pain during manipulation. In certain instances, a simple extension and traction, as, for example, hanging cast on the arm (Fig. 9) or a Balkan frame, Thomas splint, etc., on the lower extremities (Fig. 14), will lead to satisfactory results. The general principle, however, remains, that in order to prevent demineralization of the bones from continued disuse, active or passive motion of the affected limb is permitted as soon as this is thought reasonably safe.

SUMMARY AND CONCLUSIONS

The present study is based on a material of 33 cases of giant-cell tumor of bone treated from 1923 to 1935, inclusive. In a first series, curettage and postoperative irradiation were used, with 3 cases eventually coming to amputation and one resulting in sarcomatous degeneration and death. In a second series irradiation alone was employed. Among 18 cases so treated, good local result was obtained in every instance. Two patients died later from incidental causes.

In general, the trend of therapy has been toward conservatism. Following the contribution of the Bone Tumor Registry of the American College of Surgeons, the more radical surgical procedures, such as resection or amputation, were replaced by curettage and swabbing of the bony cavity with escharotics. Then irradiation was instituted, in the beginning as an adjunct to surgery and later as a primary method. Here again during the first few years, more radical procedures with large doses and short intervals between the series were employed. When, however, clinical experience showed that severe reactions were undesirable, the doses were made smaller and the intervals between the series longer. This coincided with the change from the histogenetic interpretation of the lesion as a true blastoma to that regarding it as a product of inflammation and repair.

Primary radiation therapy is more advantageous than surgery (1) since it does

not lead to infection, (2) since reossification of the tumor-destroyed area occurs more rapidly and more completely, and (3) since, with the proper technic, malignant degeneration is obviated.

Apart from these considerations, radiation therapy is indicated as a primary method of approach because of its simplicity and complete innocuousness to the normal structures. No time is wasted in applying it first, inasmuch as the refractory cases always can be subjected to surgery later.

In administering radiation therapy two factors must be taken into consideration: (1) the adjustment of the technic to the newer concepts of histogenetic interpretation and (2) the special management of the patient during the period of reossification.

Irradiation is usually carried out with 200 kv. equiv. (1 mm. Cu forefilter). The initial dose represents 70 per cent SUD and is reduced at the rate of 10 per cent SUD per series subsequently. The full series is given in one day, several fields being used for the purpose of cross-firing. The treatment is repeated within two months and at gradually spaced intervals thereafter.

The duration of irradiation may extend over a period of two years, until complete reossification of the destroyed area is obtained. The ossifying capacity of the bone varies, among other things, with location and especially with age.

Some remarkable results have been observed in children with considerably smaller doses.

The general management of the patient concerns itself chiefly with the protection of the weakened structure of the affected bone during the process of healing. A limited use of the limb is allowed to prevent too much demineralization from disuse.

The satisfactory final results obtained both locally and *quoad vitam* justify the recommendation of radiation therapy as the method of choice in all giant-cell tumors and related cystic conditions of bone.

We are indebted to Dr. P. F. Morse, pathologist of Harper Hospital, for the pathologic guidance upon which the underlying principles of therapy were based.

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Harper Hospital, Detroit

DISCUSSION

James A. Dickson, M.D. (Cleveland, Ohio): I appreciate very much the opportunity of discussing the paper of Dr. Leucutia, Dr. Witwer, and Dr. Belanger. Their study has involved extensive work and careful consideration of their material. I cannot agree, however, with their conclusion that primary radiation therapy is more advantageous than surgery, largely because their surgical reports do not include what is now accepted as the most ideal surgical treatment for this type of case. It is true that the old method of curettement and the use of escharotics and packing of the cavities with bismuth paste often resulted disastrously, but the surgical treatment of these cases by meticulous curettement and the filling of the cavity with bone chips has not been given the consideration that I feel it should have in arriving at their conclusions. I do not believe that we obtain a true evaluation when we compare modern x-ray technic with surgical procedures that were carried out in the twenties. I do not feel that we are going to get a true picture or a determination of the most suitable type of treatment until we have had an opportunity to compare the results of modern surgery with modern x-ray therapy.

I have had cases which have been cured by x-ray therapy, especially giant-cell tumors of the spine and pelvis, but with x-ray therapy alone the results in the long bones have been disappointing. Since there seems to be a distinct difference in the radiosensitivity of these tumors, it is possible that when we have studied them further we may find that some of them will be best treated by x-ray, and some by surgery. To date, however, it is my experience that, where possible, thorough curettement, with the use of bone chips, has proved efficacious and the results have been more effective and the length of disability definitely lessened by the adoption of this procedure. When the tumor does not respond to x-ray treatment, the operation is made much more difficult due to the devitalization of the unaffected bone and the surrounding tissues, thus definitely diminishing the rate of repair.

I wish to draw your attention to three cases which illustrate the points I have emphasized. The first patient was a young man in his early twenties, who had had extensive x-ray treatment for one year for a giant-cell tumor of the lower end of the femur. In spite of treatment the tumor had increased in size and was extremely painful. The cavity was thoroughly curetted and filled with bone chips from the tibia. X-rays showed, at the end of a year postoperatively, complete healing of the cavity. This demonstrates the advantage of the use of bone chips over x-ray therapy alone.

The second case was a cystic tumor in a child eleven years of age. It was thoroughly curetted and the cavity was filled with bone chips. Roentgenograms six months postoperatively showed complete regeneration, which we had not been able to procure in a similar time by x-ray therapy.

The third case was one of giant-cell tumor involving the lower end of the radius in a girl twenty-two years of age. In spite of x-ray treatment over a period of six months the tumor had gradually increased in size. It was then felt advisable to do a complete excision of the lower end of the radius, as it was believed that the tumor had progressed too far for simple curettement. The results from massive bone grafts in this case proved effective and satisfactory.

In both the case of the tumor in the lower end of the femur and that in the radius, it was our feeling that if surgical procedures had been adopted earlier, much time and disability could have been saved. I feel strongly that surgery still has a distinct place in the treatment of giant-cell tumors.

Dr. Leucutia has shown excellent results by radiation therapy and there should be further statistical studies such as have been presented before final conclusions are drawn as to the ideal method of treatment.

Rollin Howard Stevens, M.D. (Detroit): In some of the cases presented by Dr. Leucutia as being treated by radiation alone there were evidences of surgery. Fractures had occurred. Is not a fracture a surgical condition? And do not fractures exert some influence in the cure of these diseases? I think that should be taken into consideration.

Maurice Lenz, M.D. (New York, N. Y.): I should like to ask Dr. Leucutia how frequently in his experience tumors around the knee joint having the roentgenographic appearance of giant-cell tumors prove to be osteogenic sarcomas? Because of the danger of overlooking an unrecognized osteogenic sarcoma, our surgeons at the Presbyterian Hospital are now operating on all giant-cell tumors in this location.

R. R. Newell, M.D. (San Francisco, Calif.): I am very enthusiastic for irradiation of the giant-cell tumors and I think they do best if not treated too heavily. I believe we need courage in diagnosis, restraint in treatment, and patience in waiting for results. Eight months or a year after a proper dose of x-rays might not be too long to wait.

I agree with Dr. Lenz that it is dangerous to make a diagnosis by clinical and x-ray methods of a presumptive giant-cell tumor at the knee, since it is impossible to be sure it is not an osteogenic sarcoma. But is it not true that with osteogenic sarcoma in a knee, the size of any we have seen on the screen, there is desperately small chance of saving the patient's life by amputation? If so, it is but a small chance we are asking him to take (if our diagnosis be wrong), in order to achieve a better orthopedic result if we be right. For I am convinced that with successful x-ray treatment there is less chance of leaving a severe deformity, especially in the case of a large tumor near a joint. Such a one is likely to cave in after curettement.

Traian Leucutia, M.D. (*closing*): I regret that, because of the shortness of the time, I could not read the entire manuscript as it will be presented for publication. Several of the points raised are expounded in the manuscript in detail. In closing, therefore, I shall confine myself to more general statements.

I do not wish to convey the impression that surgery is of no value, because I know that some very splendid results have been published; there is no question of that. Our idea in presenting this group was that the publication of the late results in an additional series of cases might contribute, in whatever small measure, to the further elucidation of the relative value of radiation therapy in benign giant-cell tumors.

As concerns the question of Dr. Lenz: There is the possibility that an occasional osteogenic sarcoma, especially in the region of the knee, may be wrongly diagnosed as a giant-cell tumor; but in such instances the sarcoma is usually of the osteolytic or telangiectatic type, so that here, too, irradiation is preferable to surgery. If there is some doubt about the diagnosis, one should take a biopsy and proceed accordingly.

The reactions observed in the early days of radiation therapy have been vividly described by Herendeen. They were the result of large doses of 130 or 150 per cent SUD, whereas, at the present time, only moderate doses are being used, starting with 70 per cent SUD in adults and 50 per cent SUD in children, and gradually less thereafter. This answers the point raised by Dr. Dickson, that operation is difficult after irradiation. The dosage applied really is so small that no damage results to the normal structures.

Roentgenologic Considerations in the Diagnosis and Treatment of Primary Malignant Bone Tumors[1]

WILLIAM EDWARD HOWES, M.D., and SAMUEL GEORGE SCHENCK, M.D., Brooklyn, N. Y.

BONE TUMORS have been classified by Dr. Willis C. Campbell (1) into two groups: first, osteogenic tumors, arising from elements needed in the formation of bone, namely the fibroblast, chondroblast, and osteoblast; second, non-osteogenic tumors, arising from tissues resident in bone, "but in no measure connected with the formation of bone." More recently, Ewing (2) presented a revised classification in a report from the Committee of the Registry of Bone Sarcoma of the American College of Surgeons. The first three classifications in this new listing include tumors arising from elements needed in the formation of bone, namely osteosarcoma, osteochondroma, and giant-cell tumor, while the last four classifications consist of tumors apparently arising from elements resident in bone, namely angioma, myeloma, lymphosarcoma, and liposarcoma. The full classification is as follows:

I. Osteogenic Series: Osteogenic Sarcoma
   1. Medullary and subperiosteal
   2. Telangiectatic
   3. Sclerosing
   4. Periosteal
   5. Fibrosarcoma
      (*a*) Medullary
      (*b*) Periosteal
   6. Parosteal, capsular
II. Chondroma Series
   1. Chondrosarcoma
   2. Myxosarcoma
III. Giant-cell Tumor Series
   1. Malignant
IV. Angioma Series
   1. Angio-endothelioma
   2. Diffuse endothelioma (Ewing sarcoma)
V. Myeloma Series
   1. Plasma-cell
   2. Myelocytoma
   3. Erythroblastoma
   4. Lymphocytoma
VI. Reticulum-cell Lymphosarcoma
VII. Liposarcoma

[1] Presented before the Radiological Society of North America, at the Twenty-fifth Annual Meeting, Atlanta, Ga., Dec. 11–15, 1939.

This communication is presented as an analysis of 40 cases of proved primary malignant tumors of bone which have come to the authors' personal attention. These cases were taken from the files of the Jewish Hospital of Brooklyn, Caledonian Hospital, Brooklyn Cancer Institute, and from the personal records of the authors. Each case in the series has been proved pathologically, either by biopsy or autopsy, and is classified according to Ewing's revised classification, as follows:

Osteogenic sarcoma	24 cases
Chondrosarcoma	3
Giant-cell tumor (malignant)	0
Endothelioma	5
Multiple myelomata	3
Reticulum-cell lymphosarcoma	4
Liposarcoma	0
Unclassified	1
TOTAL	40 cases

The cases have been studied from the standpoint of: (1) age and sex; (2) history of the disease, including such data as (*a*) relation to trauma and (*b*) duration of symptoms; (3) physical examination; (4) laboratory data; (5) classification into pathological types; (6) clinical diagnosis; (7) roentgen diagnosis with special consideration of (*a*) its limitations and (*b*) its accuracy; (8) management or treatment; (9) end-results.

AGE AND SEX

The age range for the series is from two and a half years (Case 30, lymphosarcoma of rib) to seventy-two years (Case 34, angio-endothelioma). The average was 32.6 years. The age distribution was as follows:

Under 20 years	12 cases
From 20 to 40	16
Over 40	12

An analysis of the osteogenic series shows

that the average age of patients in this group of 24 cases is but 30.8 years, even though three patients (Cases 7, 32, 38) over fifty years of age are included. Oddly enough, two of these older patients had primary involvement of the mandible. Our only case of osteogenic sarcoma engrafted upon a Paget's lesion (Case 33) was in a patient forty-six years old.

The age incidence in multiple myeloma is higher. The range for the 3 cases is from thirty-eight to sixty; the average forty-six years.

This entire series includes 18 males and 22 females, though usually males predominate (Christensen, 3).

HISTORY

Subjectively, the chief complaint is pain, with or without the presence of swelling. Pain preceded the presence of a mass in all of the listed cases except two: Case 20 (sclerosing osteogenic sarcoma of the femur), in which a painless swelling was present for twelve months, and Case 39 (angio-endothelioma of ulna), with a painless swelling for five months. This is in conformity with Codman's (4) statement that "unless pain precedes other symptoms the diagnosis of osteogenic sarcoma is unlikely."

Trauma: Only 5 of the 40 patients attributed their symptoms to injury. In all of these the injury was a minor one, actually of only sufficient severity to draw attention to the part. A child of ten (Case 23) stated that she felt a sudden pain in her arm while being pulled by a companion on roller skates. She did not fall. Another patient (Case 39) had his arm outside a bus window and received a glancing blow. On rubbing his elbow following the injury he discovered a lump on the ulna, some two to three inches away from the point of injury. Only one patient appears to have any actual claim to disease following injury, a man (Case 31) who had worked previously as a "sand hog" under compressed air. The authors have considered the possible development of sarcoma at the site of a small air embolus. Search of the skeleton, however, disclosed no areas of aseptic necrosis, nor has it been possible to find any report on the development of bone sarcoma in men working under compressed air.

Duration of Symptoms: The average duration before treatment, as obtained from the history, was about 6.5 months, the extremes being a few days in Case 34 (angio-endothelioma) and eight years in Case 32 (osteogenic sarcoma of mandible). Thirty-three patients were first seen by us seven months or less after the onset of symptoms; the other 7 presented a history of eight months, nine months, ten months, a year, one and a half years, two years, and eight years. Only 8 patients in the series of 40 came to us within a month after onset of symptoms. Codman (4) writes that the history of patients suffering with osteogenic sarcoma seldom exceeds a year or is less than a month in duration.

PHYSICAL EXAMINATION

All patients except those suffering from multiple myeloma appeared well nourished and in good general health. A mass was usually present. Tenderness to deep pressure was usually absent. Negative physical findings do not preclude the diagnosis of primary bone malignancy.

The bones affected by the disease were as follows:

Humerus	10 cases
Femur	9
Multiple foci	5
Rib	4
Ilium	4
Tibia	2
Mandible	2
Scapula	1
Metacarpal	1
Ulna	1
Ischium	1

The more common sites of primary involvement, as revealed in this series, in the order of their frequency, are the humerus, femur, pelvis, rib, tibia, and mandible, exclusive of the diseases which show multiple foci. In the osteogenic group, the humerus was affected in 8 cases, the femur in 6, the

pelvis in 5, the rib in 3, the mandible in 2, and the scapula and metacarpal bone in one each.

LABORATORY DATA

Except for the microscopic sections, laboratory data are of little diagnostic value.

The determination of serum phosphatase may be of some help. According to Woodard and Higinbotham (5), "a high serum phosphatase is an indication of the amount of new bone formation taking place in the body or an attempt on the part of the body to form new bone, and serves as a measure of the functional rather than the histologic type of the disease."

A high myelocyte count in the peripheral blood stream is an indication of irritation of the bone marrow, and may be observed in the presence of medullary tumors, particularly those of the myelomatous group.

CLASSIFICATION INTO PATHOLOGICAL TYPES

With adequate study and consideration there should be little difficulty in fitting each case properly into the classification given above, though there are rare instances when the roentgen appearance of the tumor is atypical and the pathologists are unable to agree. In our series, this difficulty arose once (Case 33). Because of the universal changes throughout the skeleton, the roentgenologist suggested the possibility of a metabolic dyscrasia, whereas, on autopsy, the pathologist listed the case as a generalized leukosarcoma. This case represents a malignant new growth, primarily arising in the osseous system, and cannot properly be ignored in this discussion (Fig. 19).

Several cases have been eliminated from this series because of a difference in opinion regarding the exact diagnosis of the microscopic section. For instance, a man, twenty-six years old, had a tumor removed from the frontal bone, which was originally described as a plasma-cell myeloma. On reviewing the case for this communication, the following diagnoses were made by prominent pathologists: giant-cell tumor, reticulum-cell lymphosarcoma, and nonspecific granuloma.

CLINICAL DIAGNOSIS

Clinically, osteogenic sarcoma is suspected from the history, which includes the age, mode of onset, duration of symptoms, and rapidity of growth; also from the physical examination. No matter how painstakingly the above procedures are carried out, however, they are inadequate without the x-ray film and the pathological section.

ROENTGEN DIAGNOSIS

Of paramount importance in the diagnosis of primary bone neoplasms is the roentgen study. The disease often produces a characteristic image which discloses its location and extent. Differentiation from metastatic disease, benign tumors, inflammatory conditions, endocrine dyscrasias, and other clinical entities may not always be simple (see Table I).

Limitations: The roentgen study is by no means infallible and certain limitations may be considered:

(1) Metastatic Disease: The differentiation between primary bone tumors and metastatic disease is of prime importance, from the roentgenologic point of view. Kolodny (6) points out that the case which gave Codman the inspiration to institute the Bone Registry was proved later to have been a metastatic carcinoma.

(2) Osteogenic Series: The radiating spicules seen most frequently in osteogenic sarcoma of the subcortical and periosteal types are by no means pathognomonic. Anspach (7) reports them as typical in giant-cell tumors of the calvarium. They have been described in osteomyelitis, primary chondrosarcoma, meningioma, and erythroblastic anemia, and have been shown by W. Loepp (8) in Ewing's sarcoma. They are beautifully illustrated in Case 13, of our series (Fig. 1). This phenomenon has been recognized by Westing (9) in his work at the Brooklyn Cancer Institute, where he has observed it twice in

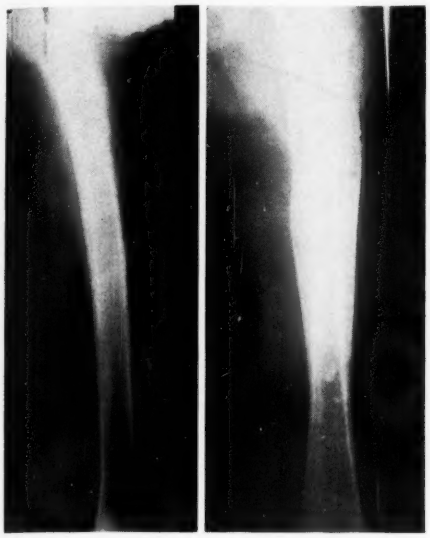

Fig. 1. Case 13: Ewing sarcoma before (left) and after (right) irradiation. Note the radiating bone spicules at right angles to the shaft of the femur (best seen on the original films).

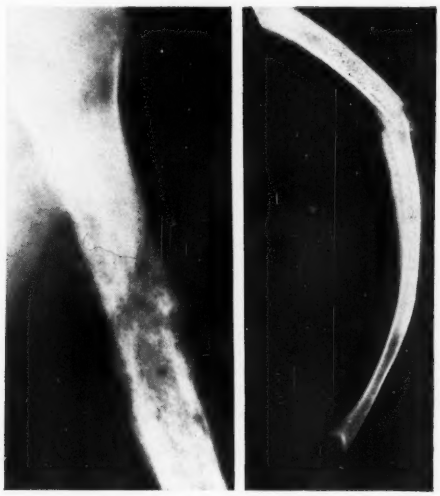

Fig. 2. Radiating bone spicules in tumors other than osteogenic sarcoma: at left, lymphosarcoma of humerus (Case 28): at right metastatic carcinoma of rib (roentgenogram of specimen) from primary carcinoma of bladder.

metastatic carcinoma secondary to a bronchogenic tumor, once in metastatic carcinoma secondary to carcinoma of the bladder, once in metastatic carcinoma secondary to a Wilms tumor, once in a local extension of an epithelioma in the skin, and once in a lymphosarcoma, primary in bone (Fig. 2).

Fibrosarcoma and tumors of the parosteal group may be extracortical and invade the cortex only by continuity. They "arise from fibrous capsules of joints and deep fasciae, often produce bone and cartilage, and may be intimately attached to the bone, but they do not originate from true bone tissue" (Ewing, 2). On the x-ray film all that will be seen is a deep soft tissue mass with possibly some destruction of contiguous bone.

(3) Osteochondrosarcoma: The benign osteochondroma can rarely be differentiated from the malignant osteochondrosarcoma until the size and extent of the tumor and its destructive and invasive proclivities make the differentiation self-evident (Case 6; Fig. 10).

(4) Giant-cell Tumor: There is no reported criterion for distinguishing between the benign giant-cell tumor and its malignant variant, except when recurrence follows surgery or irradiation or when distant metastases can be demonstrated. Even the presence of local recurrence and the spread of the tumor across joint spaces do not necessarily indicate malignancy. These lesions sometimes recur locally and spread centrifugally, but yet remain benign.

(5) Ewing Sarcoma: The diffuse endothelioma may offer considerable difficulty in recognition. It is most frequently confused with osteomyelitis. Desjardins (10) has listed the following outstanding characteristics of this tumor: "(*a*) a tendency to absorb or to destroy bone and never to produce bone; (*b*) slow metastasis; (*c*) a tendency to invade soft parts, and (*d*) a marked susceptibility to roentgen rays and radium." The fact that he uses the susceptibility of this new growth to radiation as a diagnostic criterion is admission of our limitation in making the diagnosis.

(6) Myeloma Series: It is impossible to distinguish on the film between the plasma-cell, lymphocytic, myelocytic, and

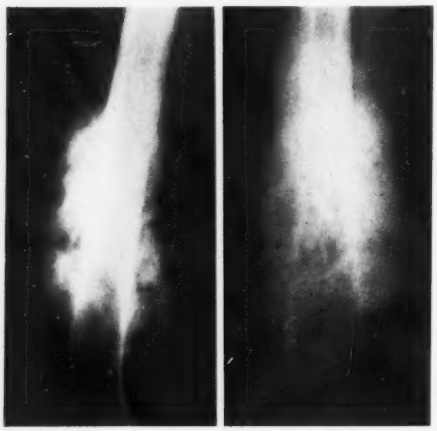

Fig. 3. Case 20: Sclerosing osteogenic sarcoma of femur, showing preservation of shaft beneath the tumor.

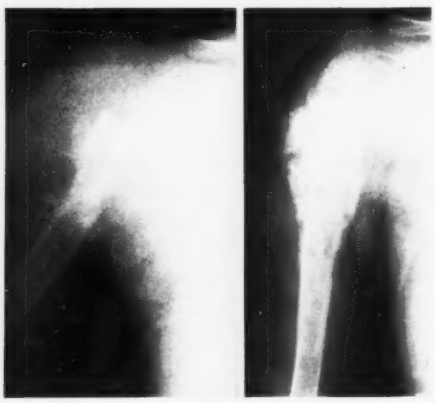

Fig. 4. Case 23: Osteogenic sarcoma in a ten-year-old girl, before (left) and after (right) irradiation. Note the soft tissue mass, radiating bone spicules, preservation of humeral shaft, elevation of periosteum, and fracture at epiphysis, before irradiation (left).

erythroblastic types, as these represent purely histologic varieties.

(7) Lymphosarcoma: The roentgen appearance of lymphosarcoma is by no means characteristic and can be easily mistaken for metastatic carcinoma or osteogenic sarcoma.

(8) Liposarcoma: This lesion is rare and, according to Coley and Peterson (11), "the radiographic appearance is not always typical and simulates most often a medullary osteogenic sarcoma."

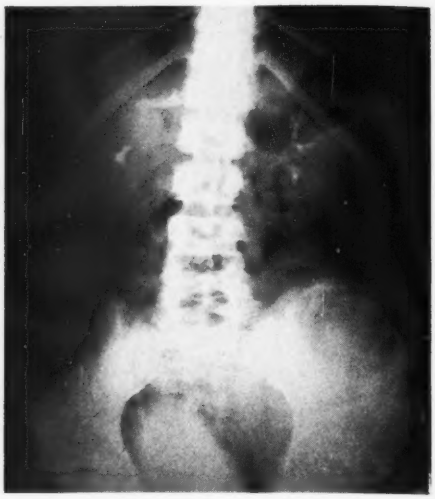

Fig. 5. Case 6: Osteogenic sarcoma of ilium. Note the large soft tissue mass filling the left side of the pelvis and displacing the bladder to the right.

Accuracy: The x-ray film often gives an accurate portrayal of the gross pathological changes. It may indicate whether the tumor is central (in the medullary canal) or peripheral (in the cortex, periosteum, and adjacent soft tissues); whether the lesion is entirely destructive to bone or is bone-forming; whether it is advancing with or without cortical or periosteal reaction; whether the growth is expansile, cystic in nature, etc.

(1) Osteogenic Series: The Registry of Bone Sarcoma has divided the osteogenic series into six subdivisions. The roentgenologist can hardly be expected to differentiate these types on the film. The most typical are the sclerosing and periosteal forms, which often have clear-cut roentgen criteria. The sclerosing osteogenic sarcoma is characterized by bone production of the patchy or cottony type, often extending far into the soft tissues. The cortical shaft is always incompletely preserved and recognizable through the dense tumor bone (Figs. 3-6).

The periosteal group is characterized by the typical sun-burst effect of radiating bone spicules (Fig. 8).

The medullary and subperiosteal group is indicated by the phenomena of destruc-

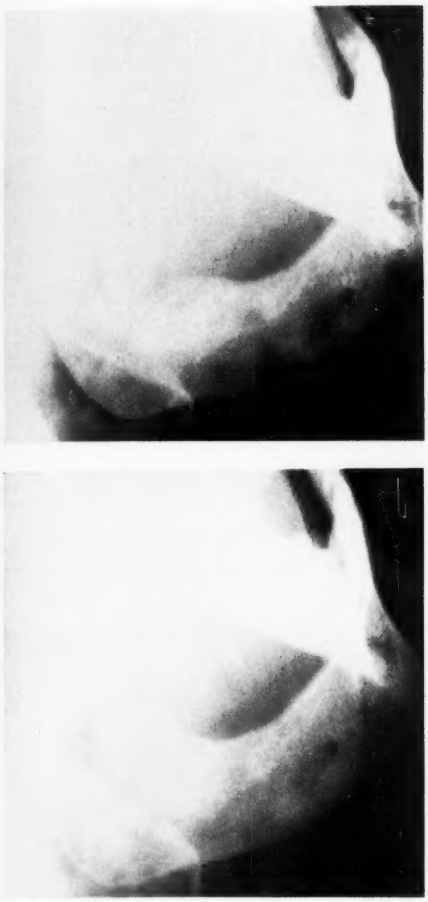

Fig. 6. Case 38: Telangiectatic sarcoma of mandible before (above) and after (below) irradiation.

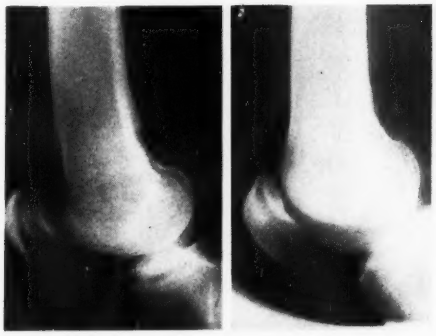

Fig. 7. Case 19: Periosteal sarcoma of femur before (left) and after (right) irradiation. Note the break in the cortex and periosteum above the condyles posteriorly, with radiating bone spicules.

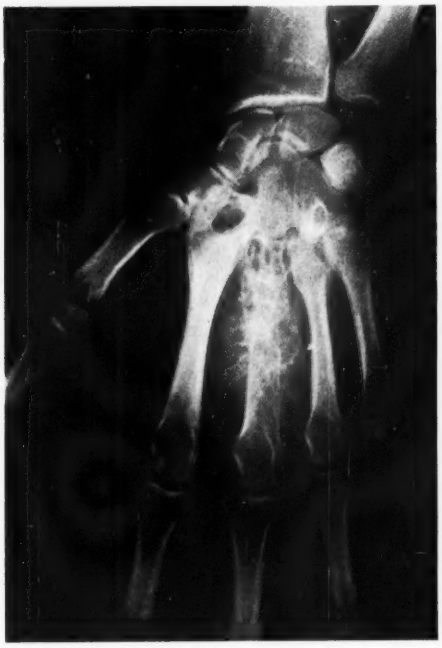

Fig. 8. Case 12: Osteogenic sarcoma of third metacarpal bone. Note the cyst or enchondroma of the lesser multangular and the base of the third metacarpal bones. These benign lesions antedated the malignant process by several years.

tion and bizarre new bone formation. The process is unusual if it does not include medulla, cortex, periosteum, and soft tissues at the same time. There is often some calcific substance within the soft tissue tumor, if radiating spicules are not present (Fig. 9). The periosteum may be elevated at the periphery of the lesion, producing the characteristic triangle of Codman (Fig. 7).

(2) Chondroma Series: Phemister (12) has shown that the chondrosarcoma presents sufficient clinical, morphological, roentgen, and microscopic characteristics to be listed as a distinct entity. He recognizes a central and a peripheral type. Tumors of the first variety tend to form large, thick-walled cystic cavities which expand the cortex, while in the second group, new bone and calcific deposits give a splotchy cottony appearance (Fig. 10).

The myxosarcoma, arising in the soft parts, affects the periosteum and cortex of

the bone by extension and invasion. The cortical destruction associated with the soft-tissue tumefaction may produce a highly suggestive appearance (Fig. 11).

ends of long bones. It perforates the shaft and invades the soft tissues and produces pulmonary metastases. It may also consist of a widespread destructive process in-

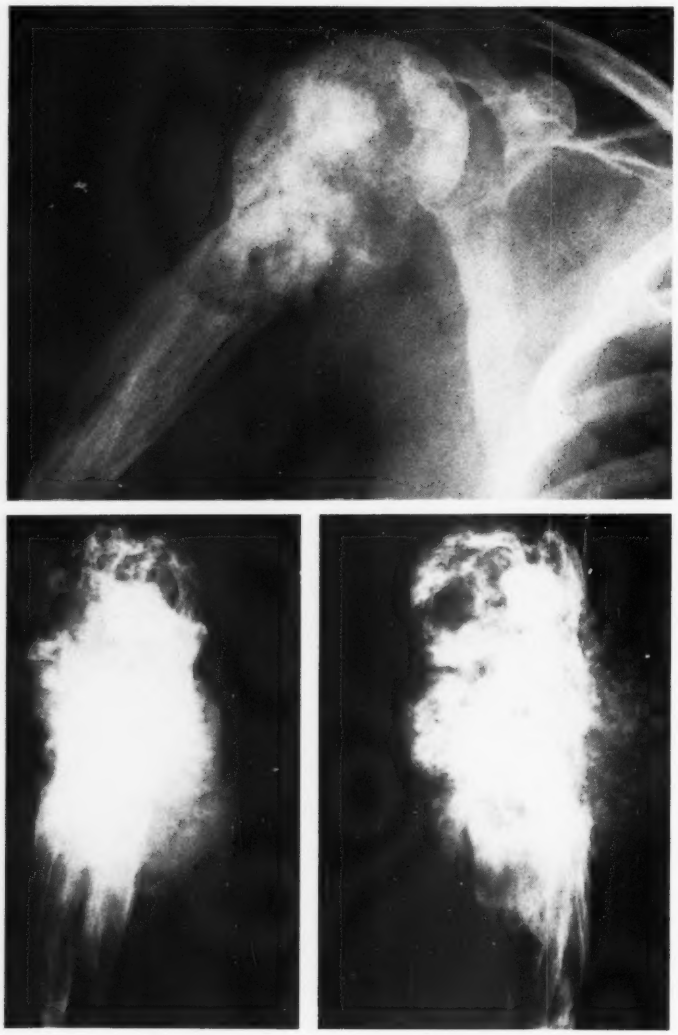

Fig. 9. Case 22: Osteogenic sarcoma arising in Paget's disease. The upper photograph shows the pre-irradiation appearance; the lower ones the operative specimen.

(3) Giant-cell Tumor Series: See above.

(4) Angioma Series: The angio-endothelioma (Fig. 12), according to Ewing (13), is an expanding single lesion involving the

volving multiple bones as in our Case 34 (Fig. 13).

Diffuse endothelioma or Ewing's sarcoma is well described by Geschickter and Maseritz (14) as "an invasive tumor, which

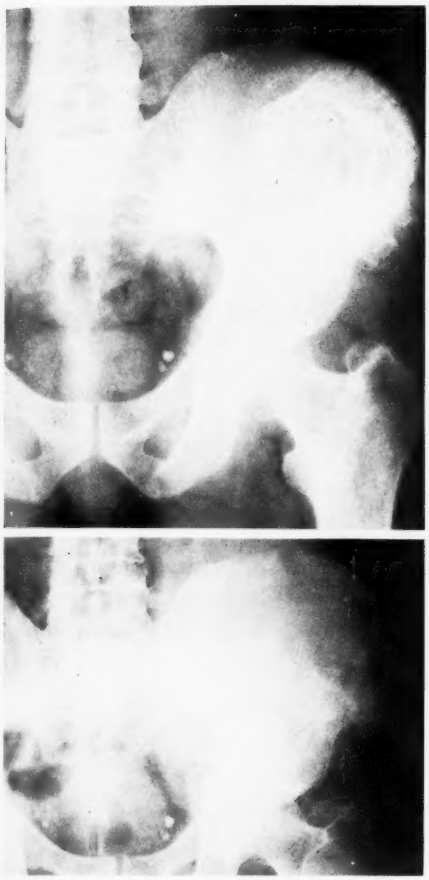

Fig. 10. Case 6: Osteochondrosarcoma. The upper roentgenogram (1934) was misinterpreted as a benign osteochondroma. The lower picture (1936) shows extension into the soft tissues with pathologic fracture of iliac crest; diagnosis, osteochondrosarcoma.

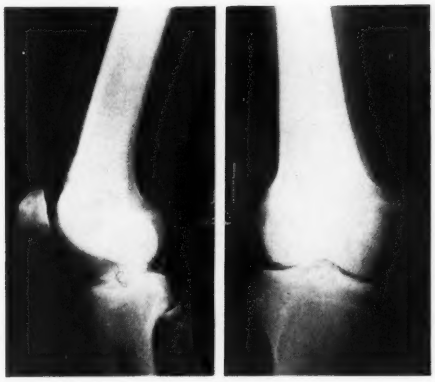

Fig. 11. Case 4: Chondromyxosarcoma. Note the break in the periosteum and cortex of the femur just above the patella. The soft tissue mass is clearly seen on the x-ray film.

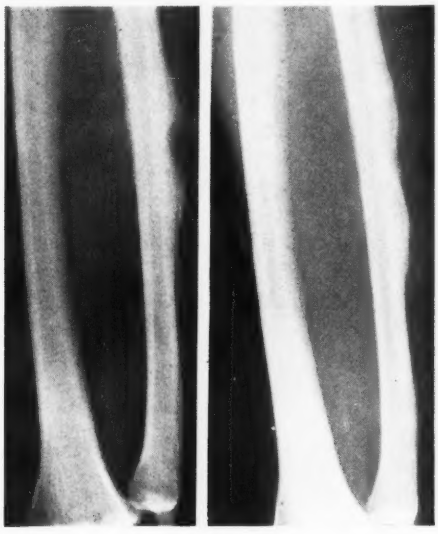

Fig. 12. Case 39: Angio-endothelioma of ulna before (left) and after (right) irradiation. Pronounced improvement clinically and radiographically.

tends to expand longitudinally along the shaft, invading the medullary cavity and destroying bone spicules in its course. In response to this invasion and destruction, new bone is laid down both subperiosteally and subendosteally. The tumor tissue possesses no properties of osteogenesis. The ratio of bone destruction and new bone formation is variable and inconsistent, giving rise to numerous roentgenographic pictures. The so-called onion-peel formation in Ewing's sarcoma is the result of multiple parallel rows of reactive bone separated by tumor tissue" (Fig. 14).

(5) Myeloma Series: Roentgen study of this series shows large and small punched-out rarefying areas, typically multiple and central and frequently distributed in the pelvis, sternum, ribs, spine, and skull (Fig. 15). Pathologic fractures are common, resulting in collapse of involved vertebral bodies (if multiple, the collapse skips several vertebrae). In the skull the multiple punched-out areas are typi-

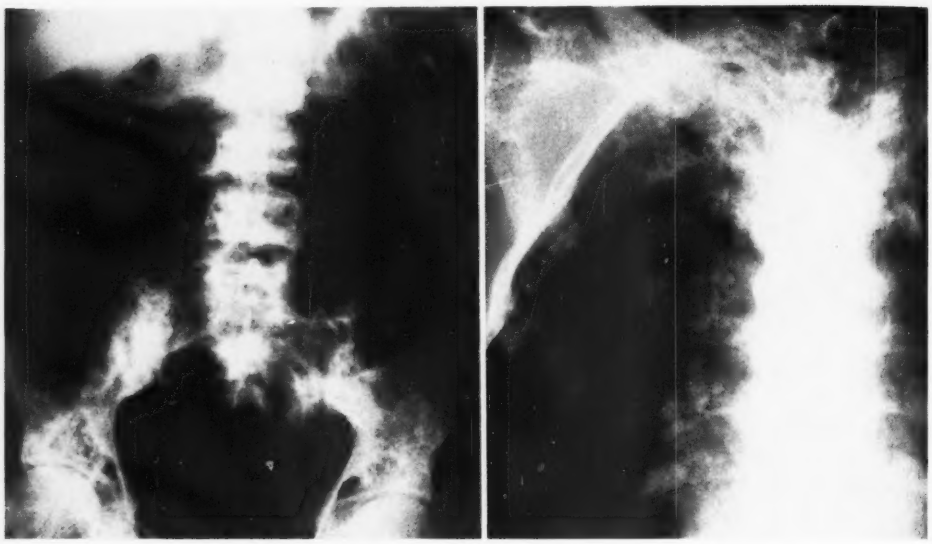

Fig. 13. Case 34: Angio-endothelioma involving the entire skeleton (Hauser, 17).

cally round except when contiguous surfaces result in flattening. The surfaces are smooth as compared with metastatic carcinoma, where the tendency is to form foci of varying shapes with markedly irregular feathered or lacy edges.

(6) Reticulum-cell Lymphosarcoma: The lesions produced by lymphosarcoma are distinctly destructive, giving a lacy, moth-eaten appearance, occurring centrally, cortically, and periosteally. While localized, the process is not discrete as in metastatic disease. Multiple bones are affected in one extremity, as in Case 8, in which the process invaded the humerus, ulna, and carpal and phalangeal bones of the right arm and hand. The disease often jumps over the intervening joints, whose surfaces may be invaded (Fig. 16).

A significant feature of lymphosarcoma is its pronounced response to radiation therapy. It is the most radiosensitive of all bone tumors. The reparative changes following treatment are frequently amazing, the destroyed bone reappearing and reforming in its entirety, as revealed in comparative roentgenograms (Figs. 17 and 18).

(7) Liposarcoma: The liposarcoma is also said to be remarkably radiosensitive. We know of no diagnostic roentgen criteria.

Accuracy of Roentgen Diagnosis in 40 Reported Cases: It is significant that in our series the roentgen impression was in agreement with the final diagnosis in 33 cases (Table I) out of a possible 39 (Case 3 was not examined roentgenologically), emphasizing the importance and accuracy of the roentgenologic survey. The failures are represented by Case 6, which was diagnosed as benign (osteochondroma for osteochondrosarcoma); Case 8, in which the roentgenograms were interpreted as metastatic malignancy but which proved to be a lymphosarcoma; Case 33, diagnosed as a metabolic dyscrasia, but shown at necropsy to be a leukosarcoma; Case 34, a reticulo-endothelioma diagnosed at autopsy, upon which no roentgen diagnosis was ventured; Case 36, a parosteal osteogenic sarcoma, the roentgenograms of which were taken postoperatively (*i.e.*, after local removal of the tumor at another institution), with negative findings; Case 39, reported as cortico-periostitis of the ulna, but which proved at operation to be an angio-endothelioma.

TABLE I. CORRELATION OF ROENTGEN DIAGNOSIS WITH PATHOLOGIC DIAGNOSIS

Case No.	Bone Affected	Roentgen Diagnosis	Pathologic Diagnosis
1	Scapula	Tumor	Sarcoma
2	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
3	Rib	Not examined	Chondrosarcoma
4	Femur	Osteomyxochondrosarcoma	Osteomyxochondrosarcoma
5	Femur	Periosteal sarcoma	Osteogenic sarcoma
6	Ilium	Osteochondroma	Osteochondrosarcoma
7	Humerus	Sarcoma	Osteogenic sarcoma
8	Tibia	Metastatic malignancy	Lymphosarcoma
9	Femur	Sarcoma	Periosteal sarcoma
10	Osseous system	Metastatic disease or myelomata	Plasma-cell myeloma
11	Rib	Sarcoma?	Osteosarcoma
12	Metacarpal	Osteogenic sarcoma	Osteogenic sarcoma
13	Femur	Ewing's sarcoma	Ewing's sarcoma
14	Osseous system	Multiple myeloma	Plasma-cell myeloma
15	Femur	Ewing's sarcoma	Ewing's sarcoma
16	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
17	Rib	Sarcoma?	Osteogenic sarcoma
18	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
19	Femur	Periosteal sarcoma	Periosteal sarcoma
20	Femur	Sclerosing osteogenic sarcoma	Sclerosing osteogenic sarcoma
21	Osseous system	Multiple myeloma	Plasma-cell myeloma
22	Humerus	Paget's disease with sarcomatous changes	Paget's disease and osteogenic sarcoma
23	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
24	Ilium	Osteogenic sarcoma	Osteogenic sarcoma
25	Tibia	Malignant tumor	Ewing's sarcoma
26	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
27	Ilium	Osteogenic sarcoma	Osteogenic sarcoma
28	Humerus	Sarcoma	Lymphosarcoma
29	Humerus	Sarcoma	Lymphosarcoma
30	Rib	Neoplasm	Lymphosarcoma
31	Femur	Malignant bone tumor	Osteogenic sarcoma
32	Mandible	Osteogenic sarcoma	Osteogenic sarcoma
33	Osseous system	Metabolic dyscrasia	Leukosarcoma
34	Osseous system	?	Angio-endothelioma
35	Humerus	Osteogenic sarcoma	Osteogenic sarcoma
36	Femur (knee)	Negative	Capsular parosteal osteogenic sarcoma
37	Ilium	Osteogenic sarcoma	Osteogenic sarcoma
38	Mandible	Sarcoma or adamantinoma	Osteogenic (telangiectatic) sarcoma
39	Ulna	Cortical periostitis	Angio-endothelioma
40	Ischium	Malignant tumor	Osteogenic sarcoma

MANAGEMENT OR TREATMENT

Twelve years ago Kolodny (6) wrote: "The vast accumulation in recent time of technical knowledge and the advance in the application of radiation, roentgen ray, and radium, in the therapy of malignant tumors in general required a re-evaluation of all accepted principles and traditions, as far as the therapy of bone sarcoma is concerned. Even now clinicians are inclined to think that if a bone sarcoma is operable, there is an indication for surgical treatment without losing time in radiation, while for radiation treatment only the inoperable cases are to be left. That this view is obsolete and not in accord with the disappointing results in radical surgical treatment is obvious. It is true that no astounding success has as yet been achieved by radiation therapy of malignant bone tumors and that most results here are unsatisfactory; however, the evidence on hand is encouraging, and radiation is as legitimate a therapeutic procedure in bone sarcoma as radical surgery."

The writers regard Kolodny's words as prophetic and believe radiation therapy will be further accepted, alone or in conjunction with surgery, as of outstanding importance in the treatment of primary malignancy of bone.

Biopsy: At the Memorial Hospital biopsy appears to be performed almost routinely in bone tumors (Pack, 15) and it seems to be the feeling of workers there that the trauma incidental to obtaining adequate material for examination is of minimal consequence, as compared to the necessity and advantages of an accurate pathological diagnosis.

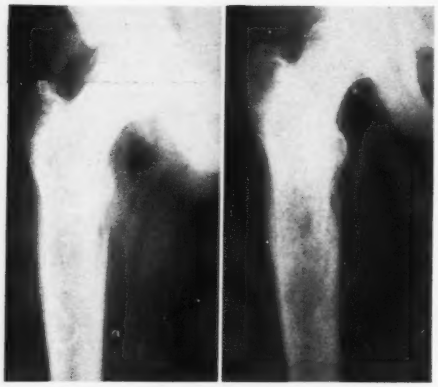

Fig. 14. Case 13: Ewing sarcoma of femur before (left) and after (right) irradiation. Note the radiographic improvement as evidenced by increased deposition of bone in the affected areas.

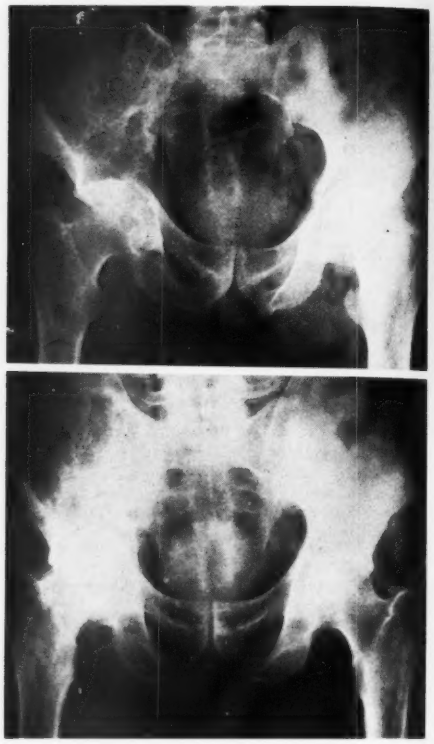

Fig. 15. Case 14: Multiple myeloma before (above) and after (below) irradiation. Observe sclerotic reactive new bone (post-irradiation), especially in right half of pelvis.

Brailsford's (16) assertion that every patient with unquestionable sarcoma he had seen, "on whom a biopsy was performed, died within a few months" is contrary to the writers' experience. Nor can we agree with Brailsford when he suggests immobilization of the affected part and follow-up x-ray examination when a positive roentgen diagnosis cannot be made in the original films. We feel that the danger of delay in therapy outweighs that of spread due to the trauma of biopsy when preparation is made for immediate adequate treatment, surgical and radiological, should the lesion prove to be malignant.

Coley's Toxins: The authors have tried Coley's mixed toxins, but to no avail, in two cases, Case 25 (Ewing's tumor of tibia with widespread metastases) and Case 14 (multiple myeloma with extensive involvement). This experience is, we appreciate, insufficient for a definite stand as to the value of this therapeutic agent.

Procedure and Technic: The procedure generally employed by us is to obtain an incisional or aspiration biopsy before initiating therapy. If the local lesion is excised, postoperative irradiation is employed. In several cases, preoperative radiation therapy was instituted, followed preferably in six to eight weeks by excision or amputation.

The following factors were used in the irradiation of the patients in this series: 200 kv., target-skin distance 30–50 cm., filters equivalent to the half-value layer of 0.5 to 2 mm. copper. The lesion is cross-fired through 2 to 4 ports, depending on the thickness of the part, each port being larger than the actual size of the neoplasm as revealed in the roentgenogram. These areas vary from 8 × 10 cm. to 12 × 20 cm. A dose of 200 to 300 roentgen units (measured in air) is administered daily to each port (not exceeding a total of 600 r per day), for a total of 2,000 to 3,000 r to each field (not to exceed 6,000 in the estimation of the tangential and emergent beam).

END-RESULTS

Even though more and more five-year cures have been recorded, both with surgery alone and surgery combined with radiation therapy, Brailsford (16) writes that

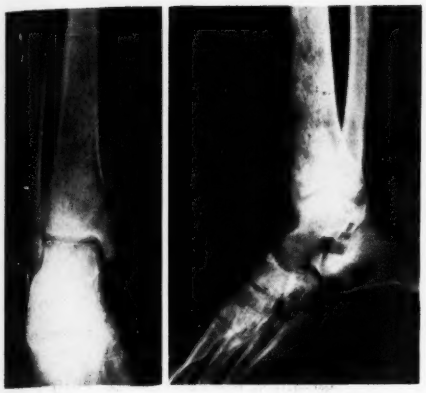

Fig. 16. Case 8: Reticulum-cell lymphosarcoma of the tibia and tarsal bones.

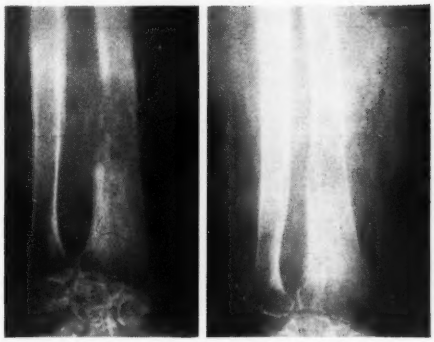

Fig. 17. Case 28: Reticulum-cell lymphosarcoma before (left) and after (right) irradiation.

only 5 per cent of some 1,500 cases registered in the Registry of Bone Sarcoma are arrested at the end of five years, and believes this number "to be within the margin of error in diagnosis." Desperate as the situation appears in the reported results, however, it is not entirely hopeless.

In our series, 5 patients (Cases 20, 27, 33, 34, and 40) received no specific form of treatment. In Case 20 therapy was refused. Two patients (Cases 27 and 40) were too ill to be moved to the roentgen department, and in Cases 33 and 34 the correct diagnosis was made only at necropsy. This leaves 35 patients who received surgery or irradiation or both. Tables II to VII epitomize the end-results in this group.

(1) *Excision:* In Case 36, a parosteal osteogenic sarcoma of the knee, two excisions of the tumor had been done before the patient was seen by us. At the time of admission to the hospital, advanced pulmonary and pleural metastases were present.

(2) *Amputation:* This was the sole method of treatment in 4 cases (Table II). In each instance but one, the humerus was the affected part, and all the lesions belonged to the osteogenic series. Three of the patients died of general metastases. In Case 20 there has been no evidence of metastasis following amputation six months ago.

(3) *Excision and Irradiation:* One patient with a periosteal sarcoma of the femur (Case 9) had a course of high-voltage roentgen treatment followed by local excision of the malignant growth and has remained well for thirteen months (Table III). In 8 cases a local excision was followed by one or more series of x-ray treatments. Three of these patients are dead (three months to three and a half years postoperatively). The other 5 are alive from six months to three years following treatment.

(4) *Preoperative Irradiation and Amputation:* In this group of 4 patients (Table IV), all with osteogenic sarcoma, 2 are dead (two days and three months postoperative) and 2 are still well over a period of four years (one six years after irradiation).

(5) *Irradiation:* Eighteen patients received radiation therapy alone (Table V). Twelve are now known to be dead and one has been lost sight of. The remaining 5 are living four and a half years, fourteen, eight, three and a half, and two months, respectively, after initial therapy. Fifteen of the 18 patients showed definite improvement with irradiation, lasting from one month to four and a half years, averaging approximately eleven and a half months. Many of these patients who

TABLE II: AMPUTATION ALONE: 4 CASES

Case No. and Diagnosis	Bone Affected	Result
2. Osteogenic sarcoma	Humerus	Died 4 months postoperatively with pulmonary metastases
16. Osteogenic sarcoma	Humerus	Died 2 months postoperatively with general metastases
18. Osteogenic sarcoma	Humerus	Died 8 months postoperatively with recurrence in stump and metastasis to lungs
20. Osteogenic sarcoma	Femur	No evidence of metastases after 6 months

TABLE III: EXCISION AND IRRADIATION: 9 CASES

Preoperative irradiation in Case 9; postoperative in all others

Case No. and Diagnosis	Bone Affected	Result
9. Periosteal sarcoma	Femur	No recurrence for 13 months
3. Chondrosarcoma	Rib	No recurrence for 3 years
4. Osteomyxochondrosarcoma	Femur	No recurrence for 6 months
6. Osteosarcoma	Ilium	No improvement; died 5 months postoperatively with general metastases
12. Osteosarcoma	Metacarpal	Improvement for 3 years; died 3 1/2 years postoperatively with distant metastasis (femur)
26. Osteogenic sarcoma	Humerus	Immediate recurrence; died 3 months postoperatively with spine metastases
30. Round-cell sarcoma	Rib	No recurrence 8 months postoperatively
38. Osteogenic (telangiectatic) sarcoma	Mandible	Symptom free for 8 months followed by metastases to spine and pelvis
39. Angio-endothelioma	Ulna	Symptomless for one year

TABLE IV: PREOPERATIVE IRRADIATION AND AMPUTATION: 4 CASES

Case No. and Diagnosis	Bone Affected	Result
5. Osteogenic sarcoma	Femur	No recurrence for 4 years
7. Osteogenic sarcoma	Humerus	Improvement for 8 months following irradiation Died of metastases 3 months after amputation
22. Paget's disease and osteogenic sarcoma	Humerus	No improvement for 2 months after irradiation. Died 2 days postoperatively
23. Osteogenic sarcoma	Humerus	No recurrence for 4 years

TABLE V: RESULTS WITH RADIATION THERAPY ALONE: 18 CASES

Case No. and Diagnosis	Bone Affected	Results
1. Sarcoma	Scapula	Improvement for 5 months. Died 7 months later of metastasis to lungs
8. Lymphosarcoma	Tibia	Improvement for 1 1/2 years followed by general metastases and death, 6 months later
10. Myelomata	Osseous system	Marked palliation lasting for months at a time. Died about 3 years after initial irradiation
11. Osteosarcoma	Rib	Improvement for 5 months. Died of extension, 3 1/2 months later
13. Ewing's sarcoma	Femur	Improvement for 4 months. Died of general metastases, 2 months later
14. Myelomata	Osseous system	No improvement. Died 18 months after onset
15. Ewing's sarcoma	Femur	Improvement for 6 months. Died of metastases to chest, 6 months later
17. Osteosarcoma	Rib	Marked improvement for 1 year. Recurrence and death from metastases to lung and mediastinum, 4 months later
19. Periosteal sarcoma	Femur	No improvement. Died of general metastases 4 months after irradiation
21. Myelomata	Osseous system	Marked improvement. Still alive 4 1/2 years after onset
24. Osteogenic sarcoma	Ilium	Improved when last seen, one month after therapy
25. Ewing's sarcoma	Tibia	Improvement for 7 months. Died of generalized metastases 1 month later
28. Lymphosarcoma	Humerus	Improvement for 5 months. Died of pulmonary and osseous metastases 1 month later
29. Lymphosarcoma	Humerus	Improvement for 8 months. No further follow-up
31. Osteogenic sarcoma	Femur	Improvement for 3 months. Pains relieved to present, 14 months after onset
32. Osteogenic sarcoma	Mandible	No improvement. Died of general metastases 11 months after irradiation
35. Osteogenic sarcoma	Humerus	Immediate improvement following x-ray therapy given two months ago
37. Osteogenic sarcoma	Ilium	Pronounced clinical and radiographic improvement for 2 months when metastases to skull were discovered

were treated by irradiation alone had advanced disease and were considered hopeless.

Statistical Analysis of End-results: A general survey of the 35 cases receiving some form of specific therapy, such as operation or irradiation, or both, shows that 26 patients were benefited by treatment from one month to four and a half years, including the 13 alive at the present writing. This gives a present mean average of 15.4 months of alleviation of symptoms. The remaining 9 patients showed no clinical improvement from surgery or irradiation.

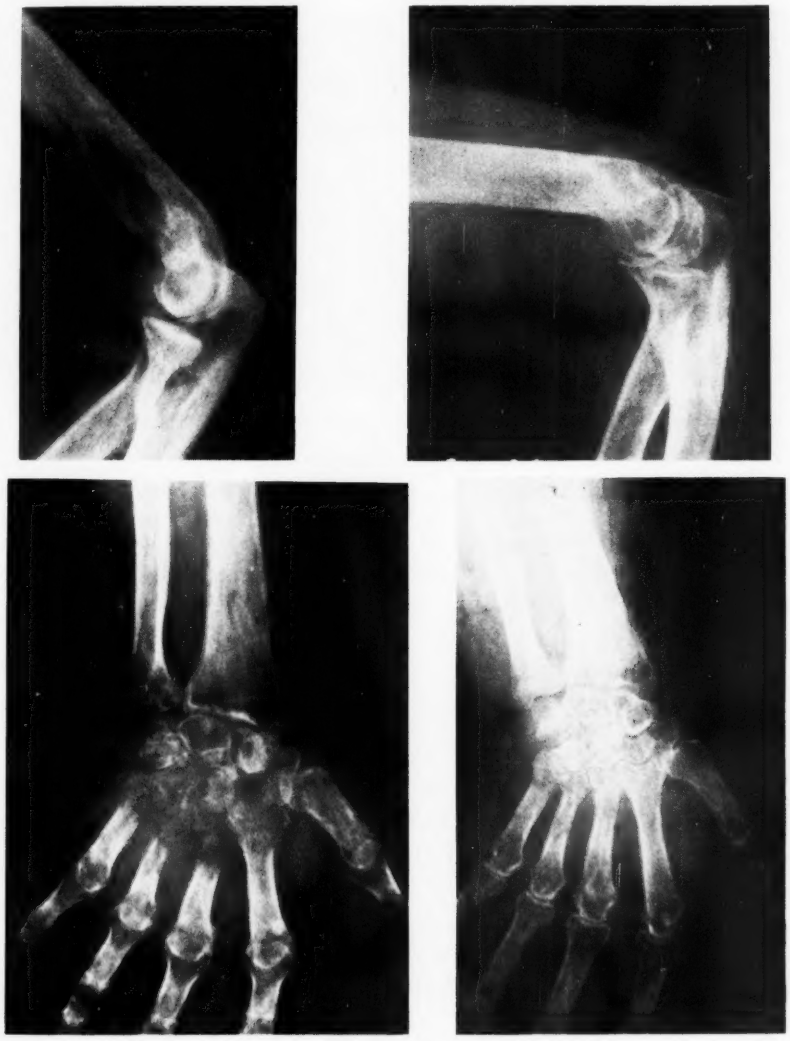

Fig. 18. Case 28: Reticulum-cell lymphosarcoma before (left) and after (right) irradiation. Irradiation resulted in pronounced clinical and radiographic improvement.

Up to the present time, 25 of this series of 40 patients are known to be dead, 2 were not followed, and 13 are living. Table VI shows the average duration of symptoms from onset to exitus for each group of

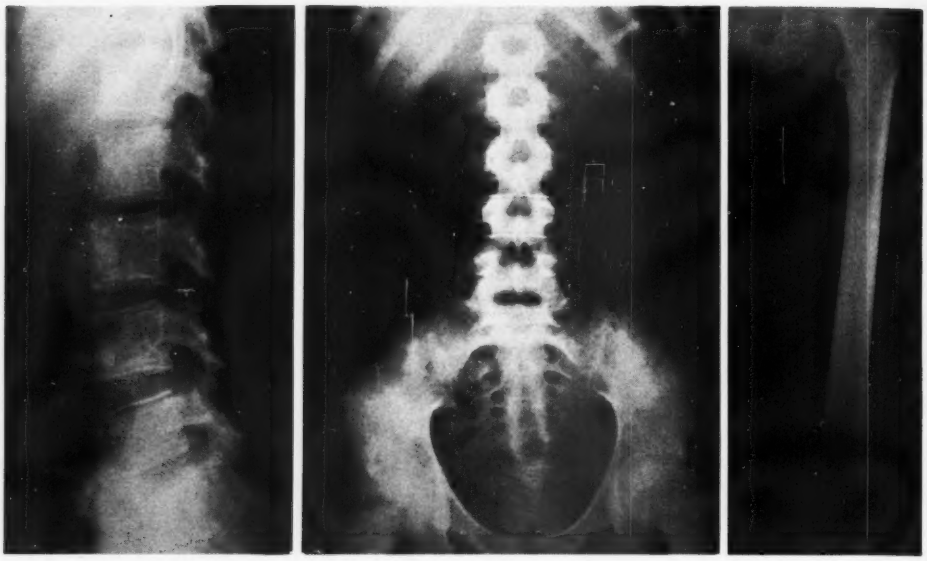

Fig. 19. Case 33: Leukosarcoma. This case could not be grouped under the revised classification. Autopsy disclosed a generalized skeletal involvement with metastases in the lung.

tumors. Patients with multiple myeloma seem to live the longest (thirty months); the shortest course is represented by the solitary case of angio-endothelioma (two and a half months). The mean for all types is fourteen months.

TABLE VI: SURVIVAL PERIOD IN 23 FATAL CASES

Type	No. of Cases	Average Duration from Initial Symptoms
Osteogenic sarcoma	16	19.2 months
Ewing's sarcoma	3	10.3 months
Multiple myeloma	2	30 months
Lymphosarcoma	2	18.5 months
Leukosarcoma	1	6 months
Angio-endothelioma	1	2.5 months
Mean Average	25	16.9 months

A study of the 13 survivors is presented in Table VII. Of these living patients there are 7 with a diagnosis of osteogenic sarcoma, 2 with chondrosarcoma, 2 with lymphosarcoma, and one each with multiple myeloma and angio-endothelioma. The present survival period, dating from the beginning of treatment, ranges from three months to six years, the mean average being twenty and a half months. Four patients are alive three years or more after the institution of therapy, as follows: Case 3, chondrosarcoma of the rib, three years; Case 5, osteogenic sarcoma of the femur, four years; Case 23, osteogenic sarcoma of the humerus, six years; Case 21, multiple myeloma, remarkable palliation from irradiation for four and a half years.

SUMMARY AND CONCLUSIONS

The analysis of 40 cases of primary malignant bone tumors, grouped according to the latest classification of the Registry of Bone Sarcoma, shows a majority of the patients younger than forty years and an average duration of symptoms before receiving treatment of about six and a half months.

The roentgen examination is shown to be of considerable importance and represents one of the surest methods of diagnosis.

An attempt has been made to summarize the characteristics of each type of tumor, including the limitations and accuracy of roentgenography. Early roentgen surveys of any unexplained pain should be immediately followed by biopsy examination in doubtful cases.

The therapeutic procedure cannot be standardized at this time. The results

TABLE VII: SURVIVAL PERIOD FOR 13 LIVING PATIENTS

Case No. and Diagnosis	Bone Affected	Treatment	Present Survival Period
3. Chondrosarcoma	Rib	Excision and postoperative irradiation	3 years
4. Osteomyxochondrosarcoma	Femur	Excision and postoperative irradiation	6 months
5. Osteogenic sarcoma	Femur	Preoperative irradiation and amputation	4 years
9. Periosteal sarcoma	Femur	Preoperative irradiation and excision	13 months
21. Multiple myeloma	Osseous system	Irradiation	4 1/2 years
23. Osteogenic sarcoma	Humerus	Preoperative irradiation and amputation	6 years
29. Lymphosarcoma	Humerus, ulna, radius, carpal, metacarpal	Irradiation	8 months
30. Lymphosarcoma	Rib	Excision and postoperative radiation	8 months
31. Osteogenic sarcoma	Femur	Irradiation	3 months
35. Osteogenic sarcoma	Humerus	Irradiation	2 months
37. Osteogenic sarcoma	Ilium	Irradiation	6 months
38. Osteogenic sarcoma	Mandible	Excision and postoperative irradiation	8 months
39. Angio-endothelioma	Ulna	Excision and postoperative irradiation	13 months

from radical surgery alone are disappointing. In this series the best results were obtained in those cases which received irradiation, usually in conjunction with surgery.

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DISCUSSION

John T. Murphy, M.D. (Toledo, Ohio): This paper, I think, is a very important one. Dr. Howes' classification, it will be noted, is solely pathological. This is in accord with the trend of the last ten years in bone tumor work in the United States. We have been led to suppose that the diagnosis of bone tumor is much easier for the pathologist than for the radiologist. The opinion among pathologists, however, does not confirm this. The difficulties there are actually just as great as those encountered by the roentgenologist.

There has been a gradual decline in the value accorded to the roentgenogram in diagnosis in the last few years, which I think is a grave mistake. The first thing the radiologist must recognize is that the usual roentgen examination of bone tumors, with anteroposterior and lateral views, is often inadequate. If the pathologist sees a slide and is not satisfied he will ask for more slides. The radiologist should examine these cases much more carefully, making stereoscopic, tangential, and other types of views to bring out all possible points to help him in his diagnosis. He should give himself every chance, and if he does, I think his average will be just as good as the pathologist's. There will be differences of opinion, of course, but a carefully followed series of cases, I think, will prove that the difference or number of differences will be small.

Robert R. Newell, M.D. (San Francisco): It seems to me that this paper has given to us much the

sort of thing which we most need—a background of clinical experience. Each of us sees few enough bone tumors so that a careful exposition by other workers of their experience is very valuable to us.

I would like to add something which neither Dr. Murphy nor the essayist mentioned: that successful or optimum treatment of these patients depends very markedly on the radiologist and the surgeon pulling amicably and sensibly in double harness. If the consultation between the radiologist and surgeon can be had before any therapy is undertaken, the chances of carrying out the treatment sensibly, it seems to me, will be much improved.

It needs to be clearly perceived by everyone concerned that radiation and surgery may fail at the same point. Thus a Ewing's tumor might be cured locally by irradiation or, even more certainly, by amputation, but under either circumstance the patient may die from remote metastases. Such a failure cannot be used as a basis for decision between the two methods of treatment. It does not dissuade me from preferring irradiation for Ewing's tumor.

Perhaps there are, in fact, reasons for trying to make up our minds what the probable diagnosis is pathologically rather than merely trying to decide whether we are dealing with a benign lesion or a malignant one. I was glad, however, to hear Dr. Murphy say that the pathologist's word is not the last one. I would say that the response to radiation often reveals more about the nature of the disease than the pathologist could determine from a biopsy.

William E. Howes, M.D. (*closing*): I want to thank the discussants for their consideration of this paper.

We have tried to bring out that it is possible to make a relatively high percentage of diagnoses according to Ewing's latest classification, even though that is a pathological classification. We have, however, made no attempt to classify the cases according to the different subdivisions. The myeloma series, for instance, is divided purely according to cell morphology and cell etiology. As far as I know these types have no typical roentgen characteristics. This is also true to a lesser degree with subdivision in the osteogenic series.

We did try to bring out in the paper the necessity for biopsy. I have a letter from Dr. George Pack confirming our view that biopsy is of minimal danger as compared to its value as a check on the diagnosis, and I have argued against Dr. Brailsford, who has recently claimed that the reaction to radiation treatment in certain bone tumors should help in the differential diagnosis. He suggested putting these patients to bed, giving them treatment, and watching them.

We feel it is very important to make the diagnosis first and then give radical treatment. This is best accomplished by the combined efforts of the radiologist and the surgeon. Our successes in these cases have apparently been due to this combination.

The Roentgenological Manifestations of Pulmonary Edema[1]

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CONSIDERING the relative frequency of pulmonary edema, the paucity of references in the literature concerning its roentgen findings is astonishing. Standard texts on roentgen diagnosis contain only brief references to this condition; the descriptions in the literature deal largely with clinical cases, and few reports of cases verified by necropsy have been presented. Furthermore, the greatest emphasis has been placed upon a classical roentgen picture. In our experience there have been numerous variations from this typical appearance which we feel should be more fully described. Many of these variations simulate other pulmonary lesions to such a degree that frequent errors in diagnosis must result. For purposes of orientation, a brief review of the pathology and of the theories of the etiology of pulmonary edema must be undertaken.

The pathology of pulmonary edema, consisting essentially of heavy, sodden lungs, with a microscopic picture of alveoli distended with serous fluid, is sufficiently well known to merit no further discussion. A distinction between pulmonary congestion and simple edema must be made. In the former there is dilatation of the blood vessels with venous stasis, but it is not necessarily accompanied by fluid in the alveoli. Eventually an accumulation of serum in the alveoli may occur as a result of congestion, in which case both will be present. Edema, however, when it is the result of other causes than circulatory failure, frequently occurs without a corresponding congestion of the blood vessels. It should also be pointed out that serum commonly accumulates in the alveoli as a result of atelectasis of the bronchial obstruction type, but in such instances the alveoli are collapsed rather than distended. The pathology of pulmonary edema and atelectasis may thus be confused, but a reasonable consideration of both the gross and microscopic characteristics of the two diseases should make the distinction possible. Likewise, the distinction between simple edema and that occurring with inflammatory processes is clearly obvious from the macroscopic and microscopic examination of lung tissue.

ETIOLOGY

The most common cause of pulmonary edema is generally considered to be left ventricular failure accompanied by a competent right ventricle which continues to pump blood into the pulmonary vascular bed. The increased pressure in the pulmonary system causes filtration into the alveoli resulting in edema of the lungs. There are, however, other causes as well, some of which have been best classified by Coelho and Ribeiro (3).

(1) Claudication of the left ventricle such as occurs with renal sclerosis, aortic insufficiency, and similar conditions.

(2) Increased pulmonary venous pressure due to mitral stenosis.

(3) Toxic substances such as neosalvarsan, iodides, and barbiturates.

(4) Neurogenic disorders.

Coelho and Ribeiro used dogs as experimental animals. They found that clamping the aorta and the pulmonary artery simultaneously did not produce pulmonary edema. If, however, the aorta alone was ligated an edema of the lungs promptly developed. An interesting observation was made from these experiments: the older dogs with considerable coronary sclerosis died before the formation of any appreciable pulmonary edema. It appears that the capillary bed of the lungs has a definite reserve capacity which must be overcome

[1] From the Department of Radiology and Physical Therapy, University of Minnesota, Minneapolis. Presented before the Radiological Society of North America, at the Twenty-sixth Annual Meeting, Cleveland, Ohio, December 2–6, 1940.

before the pressure rises sufficiently to cause edema by direct filtration. Therefore, if death occurs soon after the failure of the left ventricle, lungs relatively free from fluid will be found at necropsy.

The toxic etiology was demonstrated by the injection of silver nitrate into the saphenous vein in healthy animals. A rather marked pulmonary edema developed in all cases as a result of injury to the pulmonary capillaries, which produced a lowering of the filtration threshold. Moon and Morgan (7) elicited similar changes by the intravenous injection of histamine, proteins, and sodium glycocholate.

The neurogenic factor has been described by Farber (5). He had become interested in this field due to the coincidental postmortem findings of brain stem lesions and pulmonary edema. He did bilateral cervical vagotomies on rabbits and found edema of the lungs at necropsy; this he attributed to loss of the vaso-constrictor action of the vago-sympathetic nerves. At the time the vagotomy was performed the thyro-arytenoid ligaments fell together and some consideration was given to the possibility that the increased negative intrapulmonary pressure thus induced might be the chief factor in the formation of the pulmonary edema. Such a process, however, was discounted, since the pulmonary findings were the same after a tracheotomy tube had been inserted.

Obviously, edema of the lungs occurs as a result of nephritis or nephrosis. There is a transudation of fluid into the lungs just as there is into the other body tissues as a result of the disturbance in excretion.

The administration of large quantities of fluid intravenously or subcutaneously with resultant overloading of the circulation has also been noted, but rather infrequently, as a cause of pulmonary edema. Obviously this overloading is of a similar character to that which is produced from heart failure, such as occurs in mitral stenosis. The causative factors, however, are strikingly dissimilar; in most instances the overloading occurs in postoperative cases where transfusions and intravenous fluids are being given for therapeutic purposes. The use of large quantities of blood, glucose, or saline solutions has become a common procedure after serious operations; the value of this method of combating shock is now well known, but some of the deleterious effects may not be so apparent. That overloading the circulation may be a factor in the production of serious, even fatal, pulmonary edema is indicated by some of the cases which are reported below.

ROENTGENOLOGIC ASPECTS

Up to the present time there have been no extensive studies of the roentgenologic appearance of the lungs in pulmonary edema. There are several reasons for this. First of all, pulmonary edema is an acute condition in most instances, and it has been felt to be unwise to move the critically ill patient to the x-ray department for examination. Roentgen examinations may, of course, be made with mobile equipment, but this procedure has not often been utilized. In the second place, the clinical diagnosis is often apparent without roentgen examination. Most of the reports in the literature deal with the chronic type, such as is associated with renal disease.

The classical roentgenologic appearance has been described as a butterfly-shaped, fairly symmetrical density involving both lungs; the density is greatest at the hilum and gradually fades out toward the periphery. The apices and the extreme bases are usually clear. The density is homogeneous in nature, obliterating all structural detail of the lung. The development of the condition has not been well described, but Lelong and Bernard (6) have taken serial films in a case which was regressing. According to these authors, the density clears in a progressive fashion from periphery to hilum.

A few references to variations from the classical appearance have been found in the literature. Barjon (1) in his book, published in 1916, states that the edema may predominate on one side or be entirely unilateral. This is a general statement,

however, and he makes no direct reference to any special case. Coe and Otell (2) say that the fluid may collect in groups of alveoli, resulting in an irregular stippled density on the roentgenogram, in contrast to the usual uniform density. Werkenthin (9) describes similar variations. Roubier and Plauchu (8) encountered two cases in which the roentgenogram exhibited confluent shadows at the bases of the lungs, which they interpreted as being due to pulmonary infarcts. At necropsy, however, these densities proved to be due to a localized pulmonary edema. There is only one case recorded in the literature where the edema was localized to the upper lobes. This case was observed by Day, Sisson, and Vogt (4) following the injection of tetanus antitoxin with the production of severe anaphylactic shock. The roentgen examination was done twelve hours after the onset of symptoms and the authors conclude that the density seen on the roentgenograms may have represented only the residual portion of a previously generalized process. Since the patient recovered, necropsy verification was not obtained.

Pulmonary congestion may be differentiated by the presence of enlarged hilum shadows and dilated vessels throughout the lung fields. Simple edema of non-cardiac origin will not show these vascular changes. Massive atelectasis gives secondary signs such as elevation of the diaphragm, shift of the mediastinum, and collapse of the chest wall, which help to identify it. In the typical cases the distinction from certain types of pneumonia, pulmonary infarct, and acute miliary tuberculosis is not difficult. In the more atypical lesions such a differentiation may require repeated examination and a consideration of the clinical findings; despite these the exact diagnosis may, at times, be impossible.

REVIEW OF CASES WITH POSTMORTEM EXAMINATION

It has been our opinion that more definite conclusions on this subject might be attained by a study of cases in which the roentgen findings could be checked against the postmortem findings. With this in mind we reviewed all cases seen at the University Hospital during the past five years in which pulmonary edema was found at necropsy. During this time numerous instances of slight edema of the lungs were seen at postmortem examination, but these were not considered of any moment. There were, however, 110 cases in which the pulmonary edema was moderate to marked.

The median age for the whole group was fifty-seven years, but the variation in age is indicated by the fact that the oldest patient was eighty years old, the youngest three months.

The 110 cases observed at necropsy were classified as to primary cause of death as follows:

Carcinoma	17
Vascular	20
Renal disturbance	8
Cirrhosis and acute degeneration of liver	5
Blood dyscrasias	4
Burns	4
Septicemia	4
Miscellaneous	22
Surgical deaths	26

The group classified under carcinoma represents far advanced cases which were beyond any hope of treatment other than palliation. The miscellaneous group is large, indicating a great variation as to cause of death. In the 26 cases included under surgical deaths, the patients died within one to three days after a major surgical procedure. This last group is particularly interesting, since the majority were given large amounts of fluid either by the intravenous or subcutaneous route or both. As far as we could determine, it made no difference whether the fluid administered was in the form of a saline or a glucose solution or as a blood transfusion. In this series of patients, succumbing after operation, we have been impressed with the frequency of pulmonary edema occurring after the administration of excessive amounts of fluid; in an appreciable percentage of this group the

edema thus induced was undoubtedly a large factor in the fatality. It is notable that physical findings in most instances gave no indication of the onset of this complication. In all instances in which large amounts of fluid are being administered it would appear wise to make repeated roentgen examinations to obtain an early warning of the development of pulmonary edema.

The degree of coronary sclerosis associated with pulmonary edema in these cases was surprisingly small. The findings were as follows:

Degree of coronary sclerosis	Number of cases
0	43
1	42
2	15
3	9
Coronary thrombosis	1

The average weight of the right lung was 105 gm. more than the left. In only 15 cases was the edema more marked in the left lung. There were 5 cases in which the edema was localized entirely to one lung, in 4 cases the right lung, in one the left. In 19 cases the edema was so marked that the weight of each lung was well in excess of 1,000 gm. It is difficult to state why the findings are usually more marked in the right lung. The most reasonable explanation appears to be that hydrostatic factors play an important rôle and that most bedridden patients lie on the right side. The larger size of the right lung may also be a factor.

As will be brought out in the case reports, there were several instances of upper lobar, unilateral and even of apparent monolobar edema. In all of these there was a slight degree of edema in the remainder of the lungs, but the moderate or marked edema was well localized.

Unfortunately, in the majority of cases coming to necropsy, roentgen examinations had not been done at all, were done a considerable period of time before the necropsy, or were unsatisfactory, largely because quantities of pleural fluid were present. Despite these handicaps, 17 cases were found in which satisfactory roentgenograms had been obtained during life. In one case examination was made immediately postmortem. In another case the clinical findings were so characteristic that the diagnosis was accepted even though the patient did not succumb. It should be noted that only those cases in which pulmonary edema was unequivocally present and in which there was no evidence whatever of pneumonia, atelectasis, or other complicating process, were selected. Numerous other cases in which a complexity of processes occurred had undergone roentgen examination, but these were not considered for the purpose of this report. The various roentgen diagnoses made on these 19 patients, all of whom proved to have nothing but pulmonary edema, are listed below.

Pulmonary edema	8
Pulmonary congestion	3
Pneumonia	3
Pneumonia or infarct	1
Pneumoconiosis	1
Congestion plus atelectasis	1
Infarct plus edema	1
Bronchopneumonia plus edema	1
	19

From the numerous diagnoses shown above it can be seen at once that differing roentgen pictures may be produced by this relatively simple condition. In order to illustrate these various roentgen findings, the following cases are reported in detail.

CASE REPORTS

Case 1: An eight-year-old girl, apparently in good health until June 1939, began to have attacks of nausea and vomiting, with right frontal headaches and slight temperature elevation. The symptoms became worse and signs of cardiac decompensation appeared in November 1939. The patient was admitted on Dec. 8, 1939, with a diagnosis of hypertension on the basis of renal disease. She was markedly dyspneic and orthopneic. Physical signs were surprisingly few, only occasional and inconstant râles being heard over the bases. There was no elevation of temperature. Treatment by means of oxygen under positive pressure was attempted, but the patient struggled against the mask, and this was discontinued. Twenty-five per cent intravenous glucose, intramuscular magnesium sulphate, and digitalis were tried without avail. The patient died on Dec. 13, 1939. The roentgenograms (Fig.

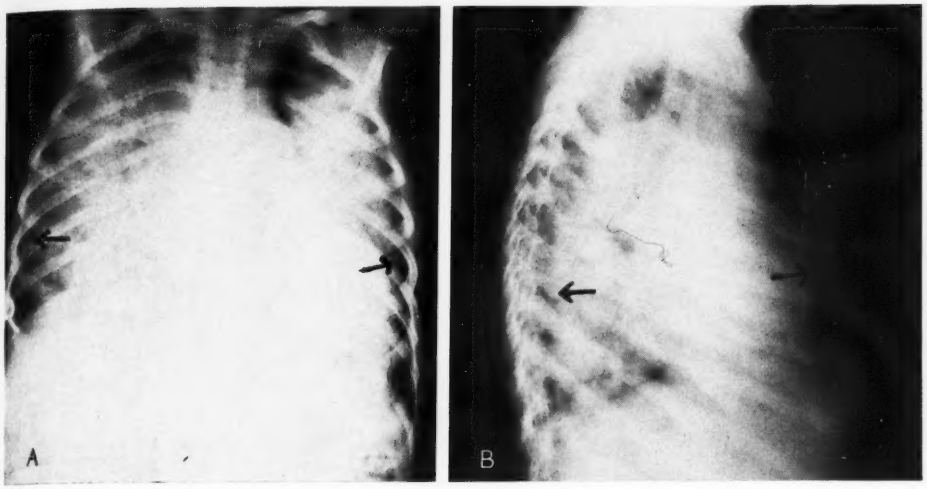

Fig. 1. Case 1: *A*. Postero-anterior roentgenogram. Typical pulmonary edema from cardiac failure. Note the bilateral, symmetrical, butterfly-shaped densities with the relatively clear periphery (arrows). While not completely homogeneous, the shadows show only slight mottling and are so dense that it is difficult to distinguish the cardiac shadow from them. Confirmed at necropsy.

B. Lateral view. Note the relatively clear periphery, both anteriorly and posteriorly (arrows), which is suggestive of the tendency for edema fluid to accumulate in the alveoli toward the center of the lung. At necropsy, however, the fluid seemed well distributed.

1-*A* and *B*) were taken on Dec. 9, about twenty-four hours after admission, and a diagnosis of pulmonary edema was made. The classical butterfly-shaped densities are seen throughout both lungs. It should be noted that the periphery of the lungs was relatively clear, as were the apices and the extreme bases. The density is of a somewhat mottled character, but on the whole is confluent and relatively homogeneous. A lateral view was obtained in this instance and it should be noted that here, too, the periphery, anteriorly and posteriorly, also appears clear.

Postmortem examination revealed a marked edema of both lungs, the right weighing 390 gm. and the left 250 gm. The heart weighed 200 gm. and showed some hypertrophy of the left ventricle. The characteristic changes of marked chronic glomerulonephritis were present in both kidneys.

Comment: This case is presented first because it illustrates the so-called typical roentgen appearance of pulmonary edema. The homogeneous, dense, bilateral shadows, sparing the apices and bases, are well shown in Figure 1-*A*. We were extremely fortunate in obtaining a lateral view, as previously no lateral views in cases of pulmonary edema have been reported. With the realization that hydrostatic factors must play a rôle in this condition, the impression has been current that in a bedridden patient, such as this child, the edema would tend to be most marked posteriorly. The lateral view (Fig. 1-*B*) illustrates, however, that the density assumes a central position, the periphery remaining clear posteriorly as well as anteriorly. On the other hand, we have made roentgenograms, postmortem, after the lungs have been removed from the chest cavity, which show the edema to extend to the surface of the lung. In the light of our present knowledge, the only explanation for this discrepancy is that the mechanics of respiration alter the peripheral distribution of the fluid in a living patient. We have found, almost uniformly, that there is a greater degree of density in the central portions of the lungs in pulmonary edema. This observation may be of some importance in the differentiation of simple edema from pneumonia, as the latter is usually peripheral in onset.

In this particular case the findings were those commonly described, and no difficulty in diagnosis would be encountered.

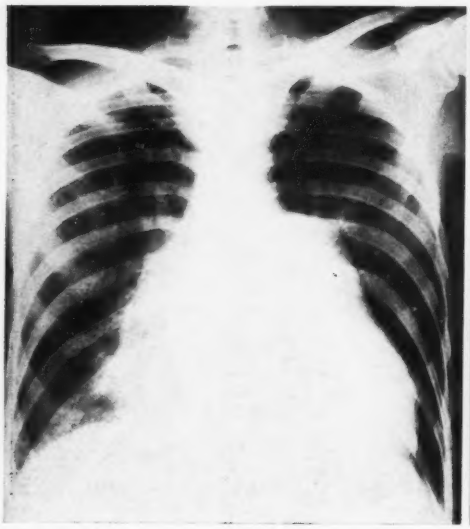

Fig. 2. Case 2: Typical pulmonary edema, in an early stage, resulting from cardiac failure in mitral valve disease. Notice the diffuse, finely mottled densities, fairly well distributed throughout both lungs, resembling, to some degree, acute miliary tuberculosis. There is an infarct in the medial portion of the right lower lobe. Confirmed at necropsy.

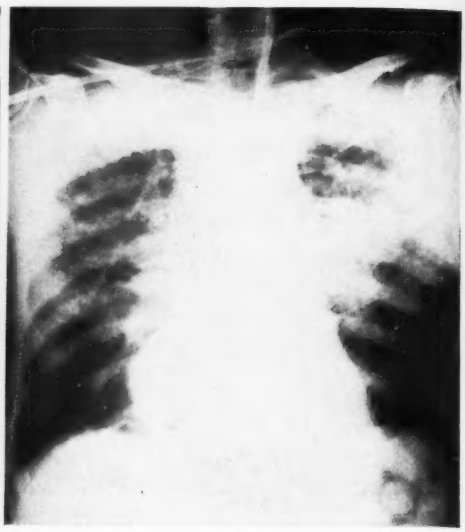

Fig. 3. Case 3: Bilateral pulmonary edema involving particularly the upper lobes, a postoperative complication due undoubtedly to overloading of the circulatory system with excess fluids. Note the resemblance to hypostatic pneumonia. Necropsy revealed no evidence of pneumonia, only edema being present. The distribution in the upper lobes was confirmed.

Case 2: A thirty-seven-year-old male was admitted to the hospital on Sept. 29, 1939. He gave a history of rheumatic fever at the age of two and one-half years with exacerbations at seven and eight years of age. The first signs of cardiac decompensation appeared at the age of thirty-three years. For ten days prior to admission there had been generalized edema, ascites, dyspnea, and orthopnea. Physical examination showed cyanosis of the finger tips and distention of the vessels of the neck. Râles were heard at both bases posteriorly. A roentgenogram (Fig. 2) made at this time shows a greatly enlarged heart of a mitral type. Marked pulmonary congestion is demonstrated, and there is early pulmonary edema, more severe on the right side. The fine mottled density throughout both lungs is notable. One density above the right cardiophrenic angle may represent an infarct. Therapy was instituted, but the patient's course was downhill and death occurred on Oct. 5, 1939.

Necropsy revealed considerable congestion and edema of both lungs, the right weighing 650 gm. and the left 470 gm. A 5-cm. infarct was present at the right base. A calcified mitral stenosis was found and the dilated left auricle was filled with a large thrombus.

Comment: Seven days elapsed between the time the roentgenogram was taken and necropsy, so that the edema most probably was more advanced at the time of death than at the time of the roentgen examination. This case is included to show the early stages of pulmonary edema. The fluid is collected in small groups of alveoli in the perivascular areas, giving the roentgenogram (Fig. 2) an irregular stippled appearance which might well be mistaken for miliary tuberculosis or capillary pneumonia. Later, when the condition progresses, the small discrete densities become confluent and produce the homogeneous density described as characteristic of pulmonary edema. Yet this early appearance is also fairly characteristic and should not involve much difficulty in diagnosis.

Case 3: A seventy-three-year-old male was admitted complaining of epigastric distress of nine months' duration. Fluoroscopic examination revealed an extensive carcinoma involving most of the stomach, and the patient was prepared for gastric resection. The operation was performed April 29, 1937. On April 30 and May 1 the patient was given intravenous fluids which were 2,000 c.c. in excess of the amount of fluid output; on May 2, 3,500 c.c.

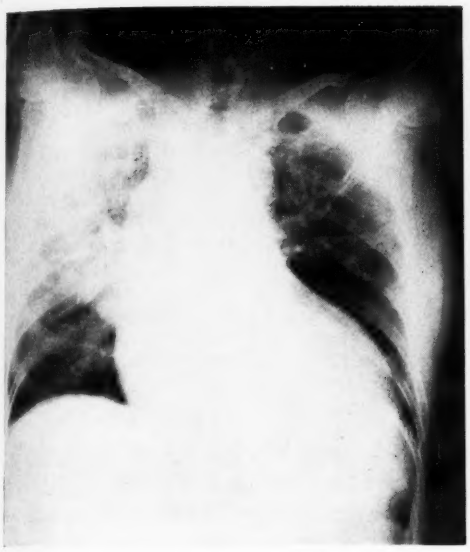

Fig. 4. Case 4: Pulmonary edema from cardiac failure. Note the extreme density around the right hilus, extending into the right upper lobe, resembling closely lobar pneumonia. There is some density in the left upper lobe as well, but of much lesser degree. The localized character of this edema is striking. Necropsy revealed only bilateral pulmonary edema

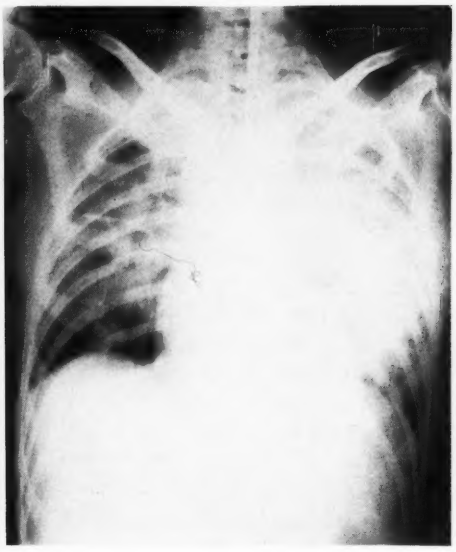

Fig. 5. Case 5: Marked pulmonary edema, occurring postoperatively, resembling bilateral pneumonia. The predominance of the findings in the left lung is striking and the rather extreme density suggests a consolidation. At necropsy bilateral pulmonary congestion and edema were found, more marked on the left.

of excess fluid were given A roentgenogram taken May 1 (Fig. 3) was interpreted as extensive bilateral, upper lobar, postoperative pneumonia. There were diffuse mottled densities in the right upper lung field and a dense irregular shadow in the left upper lung. The course was rapidly downhill and death occurred on May 3.

Postmortem examination showed markedly edematous lungs, the right weighing 925 gm. and the left 750 gm. There was no evidence of pneumonia.

Comment: This case illustrates well the fact that edema of the lungs can be largely localized to the upper lobes. The density had not yet progressed to a stage of homogeneity. Due to the stippled appearance on the roentgenogram (Fig. 3), the localization chiefly in the upper lobes, and the confluent areas of density on the left, the roentgenologist erroneously made the diagnosis of postoperative pneumonia. Overloading of the circulatory system with excess fluids was no doubt the major contributory cause of the pulmonary edema. This suggests the advisability of close check of the fluid intake and output of all surgical patients. If a great discrepancy is present in favor of the fluid intake, the roentgenologist should be alert to the fact that extensive pulmonary changes, even though not typical, may be due to an edema of the lungs.

Case 4: A fifty-eight-year-old female was admitted to the hospital on Dec. 10, 1938, with a history of severe abdominal pain for twenty-four hours. The patient had known that she had hypertension for at least three years. Physical examination revealed a triangular area of dullness over the lower half of the right lung. The clinical diagnosis was mesenteric embolism plus probable pulmonary infarction. A roentgenogram (Fig. 4) taken on the day of admission revealed a markedly enlarged heart, pulmonary congestion, plus a fairly homogeneous density in the region of the right hilus extending into the right upper lobe. This was interpreted as due to localized edema or possibly to an infarct. Emergency laparotomy was performed, and the major portion of the small bowel was resected. The patient did not rally from the operation and died Dec. 11, 1938, thirty-six hours after the roentgenogram was made.

Postmortem examination revealed a moderate edema of both lungs, the right weighing 605 gm. and the left 465 gm. There was no evidence of pneumonia or pulmonary infarction.

Comment: In this instance there was a notable localization of the edema to the right upper lung field, and in the roentgenogram taken about thirty-six hours before death little evidence of edema was made out in the remainder of both lungs. While at necropsy the edema was much more extensive, it is probable that this increase occurred during the interval between roentgen examination and death. The almost unilobar distribution of this edema is striking and indicates how closely the roentgen picture of edema may simulate that of pneumonia or pulmonary infarct. It should be emphasized that not the slightest evidence of pneumonia, infarct, or atelectasis was found at necropsy.

Case 5: A forty-six-year-old female had a Mikulicz resection of a carcinoma of the descending colon performed in October 1936. The colostomy was closed in April 1937. During the summer she had recurrent attacks of abdominal cramps, distention, and occasional vomiting. The clinical diagnosis was chronic partial bowel obstruction, and exploratory laparotomy was carried out on Nov. 11, 1937. The procedure was a tedious one, and the patient was in a critical condition at its completion. Her postoperative course was in a moribund direction from the outset, and she expired on the first postoperative day. A roentgenogram (Fig. 5) taken on the day of death was interpreted as a bilateral pneumonia; pulmonary edema was considered but believed unlikely because of the predominance of the findings in the left lung.

Necropsy revealed bilateral pulmonary congestion and edema, more marked on the left, the right lung weighing 510 gm. and the left 750 gm. There was no pneumonic consolidation.

Comment: Here we find an exception to the usual rule that edema is more extensive in the right than the left lung. The extremely homogeneous density involving the inner portion of the left lung is strongly suggestive of pneumonia, while the findings on the right side are quite characteristic of pulmonary edema. When the diagnosis was made we were not aware of the likelihood of such a discrepancy in the degree of edema in the two lungs, but it was confirmed at necropsy.

Case 6: A sixty-five-year-old female had been known to have diabetes since September 1938. She was admitted May 2, 1939, in a semi-comatose condition. Although apparently responding well to treatment, on May 4 she received 4,050 c.c. of fluid intravenously and subcutaneously and excreted only 585 c.c.; further intravenous fluids were being given on May 5, when the patient suddenly became dyspneic and cyanotic. The roentgenogram (Fig. 6) was taken at this time, and because of the unilateral density the findings were interpreted as being due to an extensive pneumonia or a very large infarct. The extremely dense shadow on the right side with the relatively clear left lung is notable. The patient died within several hours.

At necropsy a very severe edema was found involving the right lung, which weighed 910 gm. Only slight edema of the left lung was present. There was no evidence of pneumonia or infarct. A cause for the unilateral findings was carefully sought, but none could be discovered.

Comment: The extremely dense homogeneous shadow in the entire right lung without any appreciable findings in the left lung, as shown in Figure 6, is most striking. This case first aroused our interest in this subject because it seemed difficult to conceive that such a roentgen appearance could be produced by simple edema. The lungs were studied with great care at necropsy because of the questions which the roentgen diagnosis aroused and nothing but edema, almost entirely confined to the right lung, was found. It should be noted that death occurred very shortly after the roentgen examination.

Case 7: An eighteen-year-old male was admitted Nov. 17, 1937, with a one-year history of repeated frontal headaches of steady aching character. Physical examination revealed bilateral exophthalmos and bilateral diminution of the temporal visual field. Roentgen examination of the skull was indeterminate except for slight decalcification of the left posterior clinoid process.

Surgery was performed on Nov. 25, 1937, and a craniopharyngioma was found. Fluid was aspirated and a partial capsulectomy done. The postoperative course was uneventful until Dec. 2, 1937, when the patient suddenly took a turn for the worse. A roentgenogram made at this time is shown in Figure 7. The diffuse, rather mottled density confined to the left lower lung field was interpreted as atypical pneumonia. Death occurred forty-eight hours later.

Necropsy revealed an extensive extradural hemorrhage which was the cause of death. The right lung weighed 1,070 gm. and the left 900 gm. There was no evidence of a pneumonic process, the entire change in the lungs being the result of pulmonary edema.

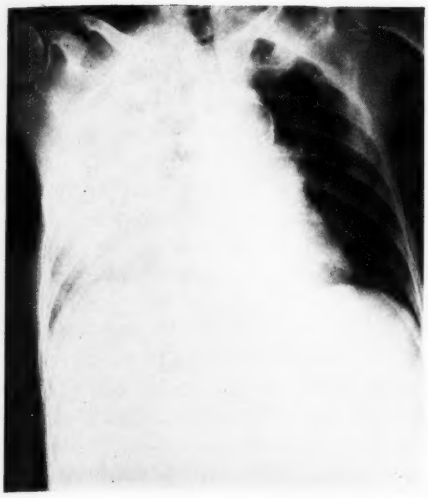

Fig. 6. Case 6: Pulmonary edema in a diabetic, given tremendous amount of intravenous fluids. Note the dense shadow involving almost the entire right lung, the left lung being practically free from pathology. At necropsy this was purely an edema of the right lung.

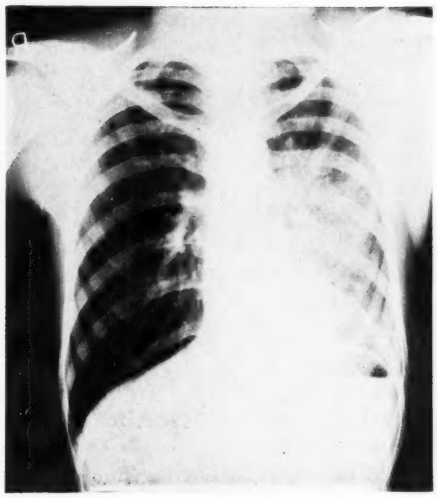

Fig. 7. Case 7: Pulmonary edema occurring eight days after brain surgery. Note the diffuse, rather mottled density involving a large portion of the left lung. There is slight elevation of the diaphragm. The right lung appears almost entirely clear. The patient died forty-eight hours later. At necropsy pulmonary edema of both lungs was found. There was no evidence of pneumonia or atelectasis.

Comment: This case is an illustration of pulmonary edema associated with brain surgery. In this instance again the roentgenogram showed only unilateral involvement. By the time that death occurred, two days later, extensive edema had no doubt developed. Unfortunately we have no roentgenograms in the interim. There is, however, no question whatever as to the aerated clear right lung in this film made forty-eight hours before the necropsy. In spite of careful search no evidence of a pneumonic process could be made out. In this instance again we have a unilateral, probably unilobar, edema in the early stages, with the eventual development of a complete edema of both lungs. In the light of the localized character of the lesion it is not astonishing that the diagnosis of pneumonia was made.

DISCUSSION

Attention should be directed to the correlation between the roentgen and necropsy findings. In those instances in which we were fortunate enough to obtain a roentgen examination within a few hours of exitus, there is a close approximation between the extent and localization of the edema as shown on the roentgenogram and that observed at the postmortem examination. This is well illustrated in Cases 5 and 6. If, on the contrary, an appreciable interval occurred between the time of roentgen examination and the time of the fatal issue, as in Cases 2, 3, 4, and 7, the postmortem findings were invariably more extensive than those exhibited in the roentgenogram. In Case 1, the findings were already so extensive when the roentgenogram was made that scarcely any further accumulation of fluid was possible. The patient's condition becoming steadily worse, there was little possibility that the necropsy would show any different picture despite the four days which intervened.

It is evident from an examination of the roentgenograms in these seven cases that pulmonary edema may simulate a number of conditions. The typical appearance, which is almost unmistakable and scarcely simulates any other pathologic process, is shown in Case 1. Case 2 illustrates the characteristic findings in the early stages

of edema, but some difficulty might be encountered in distinguishing such shadows from those of certain miliary lesions. On the whole, however, the appearance is fairly diagnostic. In these two instances no further description is needed. The majority of the cases, however, did not exhibit such a classical picture. In the remaining five cases reported, irregular distribution of the density, the tendency toward localized accumulation of fluid, the lack of homogeneity in the shadows, all tended toward errors in diagnosis. Case 3, for example, showed largely an upper lobar distribution of the density with much more marked shadows in the left upper region than on the right side. Case 4 showed a right upper lobar distribution, at least at the time of the examination, resembling very strongly the appearance of lobar pneumonia. In Case 5 the lesion is much more marked on the left side than on the right, although there is a bilateral involvement. In Case 6, both at the time of the examination and at necropsy, the edema was confined entirely to the right lung, presenting a most bizarre appearance. In Case 7 the reverse was true, the edema being confined almost entirely to the left lower lobe, although by the time death occurred a diffuse edema was actually found to be present.

The possibility that some of these localized lesions may represent massive atelectasis has been suggested. As can readily be seen from the roentgenograms, none of the collateral signs of atelectasis, such as displacement of the mediastinum, elevation of the diaphragm, or collapse of the ribs, is present. The absence of atelectasis was confirmed at necropsy.

The differentiation from pneumonic consolidation or pulmonary infarct in many of these instances is obviously difficult. The tendency for edema to give a clearer periphery is of some value. In any patient in whom the roentgen findings of consolidation are obtained, the presence of some adequate cause for pulmonary edema should dictate caution in the diagnosis of pneumonia or infarct. From our experience we believe that a definite differential diagnosis is impossible in the atypical cases. If pulmonary edema is borne in mind when acute pulmonary lesions are being studied, a correct diagnosis may sometimes be made.

It is of interest to note that in many cases of extensive pulmonary edema the physical findings are minimal. We have been repeatedly impressed by the widespread x-ray findings in patients who reveal few or no physical signs. It is possible to assign this discrepancy to two causes; first, the shallow breathing of such seriously ill individuals makes physical signs difficult to obtain; second, the tendency for the fluid to accumulate deeply in the lung rather than at the periphery may reduce the transmission of abnormal sounds.

SUMMARY

1. A study has been made of 110 cases of pulmonary edema coming to necropsy.

2. The various causes are listed. Notable among these is the administration of excessive amounts of fluid following surgical procedures.

3. Eighteen cases with adequate roentgen examinations have been studied at necropsy.

4. Seven cases are reported in detail to illustrate both the typical and atypical roentgen findings in pulmonary edema.

5. In one case lateral as well as posteroanterior views were obtained, showing the periphery of the lung to be clear posteriorly as well as anteriorly and laterally.

6. The edema was found to be localized in two cases to one lobe, in another case to one entire lung, and in others to the upper lobes alone.

7. Edema was usually found to be more marked in the right lung than the left, but in some instances the reverse was true.

CONCLUSIONS

1. Excessive intravenous and subcutaneous fluids are an important factor in the development of pulmonary edema in postoperative patients.

2. Coronary disease is not a frequent etiological factor in pulmonary edema.

3. Physical findings are often minimal in the presence of extensive roentgen findings.

4. In one case it was demonstrated that the alveolar fluid tended to collect first in groups of alveoli, later becoming confluent. This was manifested by stippled, irregular, but widespread densities in the roentgenogram.

5. The fluid tends to collect in the center of the lungs, leaving the periphery clear. As a result, a butterfly-shaped density is the classical finding.

6. The type described as having bilateral, symmetrical, homogeneous, widespread density does not occur in the majority of cases.

7. Pulmonary edema may be localized to one lobe or one lung and thus simulate pneumonia or pulmonary infarct.

8. Pulmonary edema cannot always be diagnosed when its manifestations are atypical but should be considered especially in cases of cardiac failure, renal disease, liver disease, after operations, and following transfusion reactions.

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DISCUSSION

Edgar P McNamee, M.D. (Cleveland, Ohio): This paper is an important contribution to a subject to which there are few references in the literature. Dr. Rigler and Dr. Nessa are to be congratulated for presenting it to us.

I wish to emphasize several important points. In 110 cases studied at autopsy, there were found to be 26 postoperative deaths. This means that almost 25 per cent of the deaths due to widespread pulmonary edema were postoperative. We must therefore add pulmonary edema to the list of conditions which may be found in x-ray examination of the chest following surgical procedures.

What is there in the x-ray evidence to suggest the presence of pulmonary edema and not one of the many conditions which these films would suggest to any of us? The evidence in the slides shown by Dr. Rigler makes it necessary to consider a number of pathological conditions in the lung before the diagnosis of pulmonary edema is made. In the slides where the density was widespread, the outline of the heart seemed to be more sharply defined than is usual in conditions such as atelectasis, pneumonia, and pleural effusion.

Dr. Rigler called attention to the fact that in pulmonary edema the periphery of the lung is somewhat less dense than the mid-portion and that this is true not only at the base and around the periphery in the axillary region, but also about the heart border.

Areas of the lung in which there is motion seem to have less density (edema) and better aeration than the less mobile portions. This may be a lead to the diagnosis. If we can see the outline of the heart and mediastinal structures more sharply than we would expect, with large densities in the chest, we should think of pulmonary edema.

Many questions arise. Why does edema occur in one lobe or in one lung? There is no really good explanation for this at the present time. According to the pathologist, however, the right lung usually weighs more at autopsy than the left lung. Does the right lung have more circulation than the left? Does this account for the occurrence of edema more frequently in the right lung?

I think it would be well for us to consider the pathological background for the x-ray evidence in pulmonary edema. In the roentgenogram of the average normal chest, we see linear markings, with a background of normal aerated lung. The linear markings, to a great extent, are due to the pulmonary vessels. When the blood vessels become engorged with blood, these markings naturally become more pronounced, provided the lung background is normal. As pulmonary edema develops, the blood in the vessels will leak out into the alveoli, the alveoli will be filled with blood and not air, and there will be less differentiation between the linear markings and the lung background. As the background of the normal aerated lung is replaced

by the density due to the fluid in the alveoli, the linear markings gradually fade out. With the progress of this process, more alveoli are filled with fluid and less with air and the roentgenograms show larger and more extensive areas of density. These widespread densities may simulate those of many other pathological processes in the lungs.

The difficulty of diagnosing pulmonary edema by x-ray examination is indicated by the statistics of the authors. They were able to diagnose the condition in less than 50 per cent of their cases.

This paper should stimulate all of us to be on the lookout for cases of pulmonary edema.

W. Walter Wasson, M.D. (Denver, Colorado): I had the privilege of reviewing the films shown by Dr. Rigler and I feel that we are all indebted to him for presenting them here, especially at a time when we are perhaps overlooking consideration of the cardiovascular system with fluid in the lungs.

It seems to me that there are two important points in Dr. Rigler's paper. One is that general pulmonary edema may be more prevalent than we have considered. The other is that a single lung or lobe may be involved without evidence of localized disease.

If I am to analyze such a condition, I must go back to William Snow Miller's description of the primary lobule of the lung, in which he tells us there are three systems, the ventilating system (the bronchioles and the air sacs); the vascular system (the arterioles and capillaries, particularly the latter); the lymphatic system. There is no nerve supply to the primary lobule of the lung of William Snow Miller.

We must remember that in the primary lobule there is a single cell layer of epithelium forming the air sacs, with little stroma separating the air sac from the endothelium of the capillaries; a very delicate structure, with a very delicate state of physiology. It takes little to upset the balance between the air pressure in the air sacs and the vascular pressure within the capillaries; a very slight change in either one will interfere with this balance and any such disturbance will produce the condition which Dr. Rigler has described.

Dr. Clark told us two years ago that there is no lymphatic flow without motion. There is no lymphatic flow in the lung without motion. I can understand the occurrence of general pulmonary edema with a cardiovascular disturbance and other general disturbances such as overloading of the cardiovascular system with fluids, and I can understand localized pulmonary edema with a localized condition which disturbs the balance between the air, the vascular flow within the arteries, and the lymph flow within the lymphatics, but I cannot understand how we can have pulmonary edema limited to one lobe without some local condition which interferes with this balance. That does not mean that it does not exist. I should like to have Dr. Rigler give us his concept of localized pulmonary edema without some local condition to upset this physiological balance.

Leo G. Rigler, M.D. (Minneapolis, Minnesota): I wish I could answer Dr. Wasson's last question. We discussed this matter at a seminar at the University with the physiologists and pathologists participating but got no very satisfactory answer. We can determine very little by microscopic or other examinations, about such things as the delicate balance to which Dr. Wasson has referred.

The absence of evidence at autopsy is of relatively little significance in the final analysis. We all know that there are many things which happen in the body that cannot be demonstrated at autopsy. I do think if there were any reasonable degree of atelectasis or pneumonia, we ought to be able to demonstrate it postmortem. It is conceivable, however, that there is something there that is not demonstrable by ordinary gross or microscopic examination.

I would like to emphasize one thing which I may not have made clear. In all the cases that I showed the patient died, but by no means did all the patients with pulmonary edema die. We selected cases for which autopsies were available for the sake of proving our point. We think that pulmonary edema is not infrequent after operation but patients recover from it without too much difficulty.

Compression of the Heart[1]

CLAUDE S. BECK, M.D.
Cleveland, Ohio

IN MAKING A presentation to this Society I feel that I need not attempt to discuss the radiological aspects of my subject. I know you have given considerable attention to the x-ray study of what you call constrictive pericarditis. I know that the roentgenologist has been responsible for the intrinsic lesions of the heart. It includes the vast majority of cardiac lesions. The other, smaller group consists of the extrinsic lesions, and includes those patients whose hearts are crippled by some factor just outside the heart. The extrinsic lesions of the heart deserve special consider-

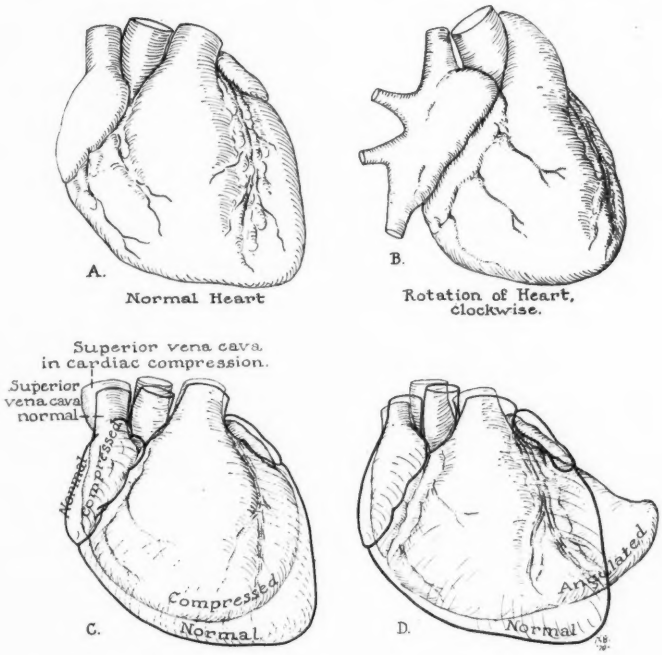


Fig. 1. Extrinsic lesions of the heart. *A*. Normal. *B*. Rotated heart. *C*. Compressed heart. *D*. Angulated heart.

the diagnosis of many instances of this lesion. I should like to present something of a supplementary nature—views gathered by a surgeon working in an experimental laboratory.

I like to divide diseases of the heart into two groups. One group consists of ation for if this outside factor can be removed by operation the patient can be cured. In extrinsic lesions of the heart we deal with a good heart together with a crippling factor. The heart is secondarily involved by an extracardiac process. This simple classification of heart disease sheds new light upon the problems of heart surgery. It helps the surgeon in the selection of patients for operation; it helps him with the operation and tells him what steps are beneficial and what steps are not

[1] From the Department of Surgery of the Western Reserve University School of Medicine and the University Hospitals, Cleveland, Ohio. Presented before the Radiological Society of North America, at the Twenty-sixth Annual Meeting, Cleveland, Ohio, Dec. 2-6, 1940.

beneficial. This classification gives something in the way of scientific order to this group of deformities and dislocations of the heart.

Extrinsic lesions lend themselves to classification and study. Indeed, they lend themselves to experimental study because they can be produced in animals. These lesions consist of (1) angulation, (2) rotation, and (3) compression. On first thought you would probably like to add

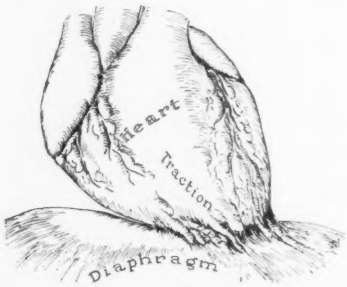


Fig. 2. Traction in the long axis of the heart is not a factor in extrinsic heart disease. Here the heart is adherent to the diaphragm.

traction as a fourth factor producing an extrinsic lesion, but as shown by experiment, traction in the long axis of the heart is not a factor in this group.

Angulation (Fig. 1-*D*) or rotation (Fig. 1-*B*) when applied to a heart experimentally produces tachycardia, a fall in arterial pressure, and a rise in venous pressure. After a few minutes these dislocations lead to ventricular fibrillation in the dog. They are equally deleterious with the human heart. If the human heart must be dislocated during an operation, we have found it advisable to carry out the dislocation for short periods of time and then give the heart a rest period. Rotation is more deleterious than angulation. These are acute dislocations of the heart. Chronic dislocations also occur. They are seen in patients with pulmonary and mediastinal tuberculosis. Angulation of the heart and mediastinum can be produced by artificial pneumothorax. Other extrinsic infections in the pericardium, mediastinum, and pleura can produce adhesions which can angulate or rotate the heart. Other mediastinal structures can be dislocated with the heart. The vagus and sympathetic nerves may be displaced with the great vessels and these displacements may produce tachycardia, dyspnea, pain, and fainting attacks. In one of my patients fainting attacks were brought on by hyperextension of the chest. Relief was obtained by bending forward. This patient's symptoms were relieved by thoracoplasty.

Traction (Fig. 2) in the long axis of the heart is not a factor in extrinsic lesions. It does not disturb the heart. It does not produce dilatation, hypertrophy, or failure of the heart. This statement has been established by considerable study in our laboratory. If a suture is placed in the apex of the heart and if traction is applied in the long axis of the heart, there is no demonstrable change in rate, arterial pressure, or venous pressure. If the heart is made adherent to the diaphragm or chest wall, and if the experimental animal is allowed to live for two or three years, no ill effects can be demonstrated. Hosler and Williams carried out this experimental study. They also analyzed material obtained from autopsy examinations, making a study of all hearts that had adhesions. These organs were separated into a group that was enlarged and a group that showed no enlargement. In each of the first group some cause for hypertrophy was invariably present, such as valvular defects, myocarditis, hypertension, etc. In no case was it necessary to assume that adhesions produced the hypertrophy. In the second group of hearts, with adhesions and without hypertrophy, no cause for hypertrophy was present.

The conclusions from these studies are of importance. They are not in agreement with the literature and they discredit the Brauer operation of cardiolysis. This operation is a thoracoplasty over the precordium designed by Brauer for the purpose of relieving the heart in its pull through adhesions upon the unyielding chest wall. If any benefit is obtained by

this operation, it must be accounted for by the correction of angulation or torsion of the heart.

Compression of the heart (Fig. 3) may be acute or chronic. Acute compression is always produced by a fluid pressure upon the outside of the heart. The fluid is usually in the pericardial cavity, but it may be in the mediastinum. A gas may compress the heart if it collects under pressure. This occurs in pressure pneumothorax. The fluid may be transudate, exudate, or blood. Acute compression is seen in stab wounds of the heart and in cases of purulent pericarditis.

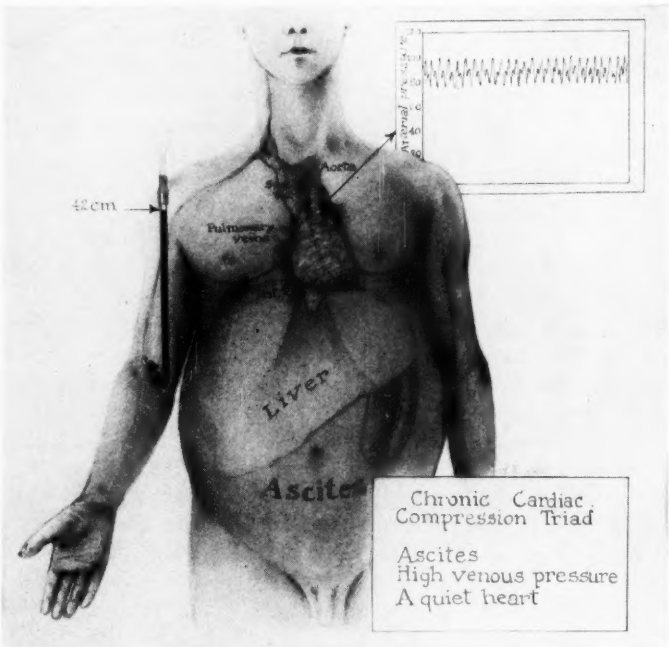
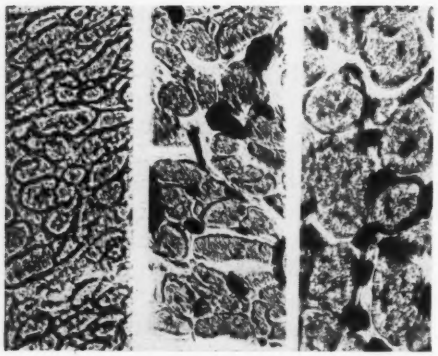


Fig. 3. Chronic cardiac compression triad.

Beck's diagnostic triad for acute compression consists of a rising venous pressure, a falling arterial pressure, and a small quiet heart. An acute compression of about 20 cm. of water is usually fatal. The venous pressure goes up to about the same pressure as that exerted upon the heart. As the compression force is increased, the venous pressure must rise in order to force blood into the heart. The compression reaches a fatal level when the venous pressure cannot equalize the pressure upon the heart. It is apparent, then, that the height to which the venous pressure can go is the factor that determines life or death in cardiac compression. The venous pressure can be raised to higher levels by intravenous administration of blood and glucose or sodium chloride solutions. This means can be used to keep the patient alive while preparation for operation is being carried out. The surgery of acute compression of the heart cannot be discussed here. It includes a discussion of stab wounds of the heart, purulent pericarditis, pericardial exudate and transudates, etc.

Chronic compression of the heart differs in several ways from acute compression. In the chronic condition the venous pressure can rise much higher than in the acute condition. The highest level in my series of cases was 45 cm. physiologic solution of



A *B* *C*

Fig. 4. *A*. Compressed heart. *B*. Normal heart. *C*. Hypertrophied heart. Notice the atrophy of the muscle fibers in the compressed heart. Same magnification in each.

sodium chloride. The high venous pressures are a protection against compression, and if they are reduced by blood-letting the patient dies. In chronic compression the arterial pressure does not fall as it does in acute compression. The arterial pressure usually measures 100 mm. mercury systolic and 80 mm. mercury diastolic. The pulse pressure is narrow and not infrequently waxes and wanes with respiration. The heart is always small and atrophic. The compressed heart cannot dilate, nor can it undergo hypertrophy. Roberts and I have demonstrated (Fig. 4) by actual measurements of heart fibers that the heart is always small and atrophic in cases of compression. The circulating blood volume increases. The amount of blood put out by the heart per unit of time is reduced. The heart plays a passive rôle in this disorder. It pumps the blood which it receives but this is less than normal. One can readily understand why the heart should undergo atrophy of disuse in this disease. In several of my patients a long time was required for the heart to overcome disuse atrophy. Up to six months may be necessary after the compression has been removed by operation.

Another manifestation of chronic compression is the water-logging of the patient. This is readily understood when one recognizes the high venous engorgement that accompanies this disease. All the veins and capillaries are filled and distended by slowly moving blood out of which the oxygen has been taken to a greater extent than from normal venous blood. The lips and nails are cyanotic. The abdomen, thorax, and soft tissues contain free fluid or edema. The heart itself is a quiet organ. The sounds are distant. There is no precordial activity. There are, of course, no murmurs because the disease is extrinsic. The intrinsic structures of the heart are not involved by the process.

Beck has described a diagnostic triad which is infallible (Fig. 3). This triad makes the diagnosis simple. Special tests are not necessary. The diagnosis need never be in error.

After the clinical diagnosis is made, the next step is to determine the anatomical lesion producing the compression. The various lesions that can produce chronic cardiac compression are pericardial compression scars (not adhesions), blood, pus, transudate or exudate in the pericardial cavity or mediastinal space, tumors of the heart or pericardium, strangulation of the heart through a rent in the pericardium, and several other rare conditions. Roentgenologic and fluoroscopic examinations are valuable in making a differential diagnosis. The x-ray manifestations of cardiac compression vary with the pathological lesion producing the compression. The one manifestation that the entire group has in common is the small, atrophic, quiet heart. It is, of course, impossible to separate the silhouette of the heart from that of the compression lesion. We have fallen into the erroneous habit of referring to the entire silhouette as that of the heart. This mistake, I am sure, has been responsible for the belief that the heart can be either enlarged or diminished in so-called mediastino-pericarditis or constrictive pericarditis. The aortic knob is flattened or obliterated. The superior cava is enlarged. The amplitude of the cardiac pulsation is always reduced. Recognition of the quiet heart is an important diagnostic point. It is important in differentiating

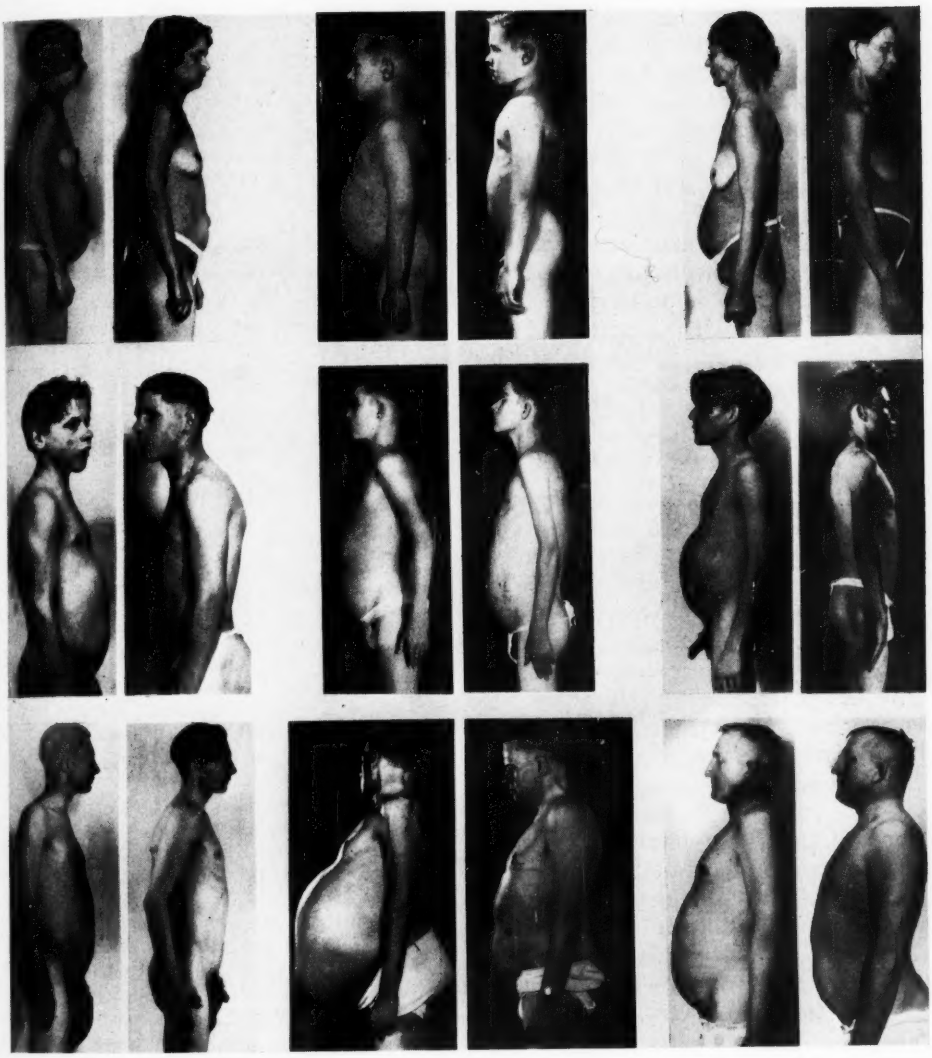


Fig. 5. Group of patients with compression scars before and after operation. Note the difference in the abdomen, nutritional state, and facial appearance.

cardiac compression from the one and only other condition that might present difficulty, namely, an obstruction to the circulation placed in the pulmonary bed.

In my experience chronic pulmonary fibrosis is the only condition that simulates chronic cardiac compression. In this condition the diastolic-systolic excursion is not reduced. We always determine whether the pulsation is reduced more on the left than on the right. It is possible to have localized compression over the right auricle and ventricle with the left ventricle relatively active. In one such case the surgical approach was carried through an incision on the right side of the sternum. Likewise, the left ventricle can be compressed to a greater extent than the right ventricle. In one specimen that I have seen, a band of calcified scar encircled both ventricles. Another specimen encountered at the operating table showed

a ring of calcium encircling the pulmonary artery. In the majority of the scar cases, a broad compression band is found well over the auricles and great vessels. It is important to divide and remove this scar formation in order to relieve its strangulating effect upon the auricles and vessels.

The presence or absence of adhesions between pericardium and heart and between pericardium, pleura, chest wall, and diaphragm is of no importance in compression of the heart. Fixation of the heart by adhesions can be determined by the roentgenologist, but this information is of no significance either for diagnosis or for determining the procedure that should be taken by the surgeon. It is not necessary for the surgeon to try to do anything about the adhesions that may form between the heart, chest wall, and diaphragm after the compression scar has been removed. Some surgeons recommend removal of the bony precordium as part of the operation; others recommend section of the left phrenic nerve. Both are unnecessary and, in fact, suggest a misconception of the physiology of the disease.

If the compression agent is fluid in the pericardial cavity or in the mediastinum, the roentgen signs are different. Here the silhouette is enormous and its contour is different. Likewise, a tumor compressing the heart gives a different silhouette, as does also a heart strangulated through a rent in the pericardium. These topics I shall not attempt to discuss.

The surgery of compression scars is a recent contribution. The operation is dramatic in its performance and scarcely less than miraculous in its immediate and remote effects upon the patient. These patients with compression scars around the heart can be cured. They can be converted from chronic cardiac invalids into perfectly healthy persons. The cure is permanent. The risk of the operation is not great if the surgeon gives every consideration to his problem. There is no other treatment except operation. Figure 5 shows some of my patients before and after operation.

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Western Reserve University

DISCUSSION

Merrill C. Sosman, M.D. (Boston, Mass.): There is little I can add to this lucid and comprehensive presentation. I should like to complement Dr. Beck on his achievements in these cases of compressive pericarditis.

My introduction to this disease occurred a good many years ago, when Dr. Wenckebach, the famous cardiologist of Vienna, was visiting the Peter Bent Brigham Hospital. I made a diagnosis of what I then called adhesive pericarditis. I asked to have the patient presented in ward rounds with Dr. Wenckebach, the following morning, with a little desire to gloat because I knew the heart was small. The only way I could see to make the diagnosis was to observe the very limited excursion of the heartbeat, and I wondered how Dr. Wenckebach, with his fingers and eyes and his stethoscope, could make the diagnosis.

I presented the case to him as one of possible adhesive pericarditis and asked him if he could make the diagnosis or rule it out. Instead of percussing or listening to the heart, he rubbed his hands together and said: "If this man has adhesive pericarditis and if it is of any significance to him, he will have an enlarged liver. Let us feel for the liver." The liver was not enlarged. Dr. Wenckebach continued: "If he has adhesive pericarditis and if it is giving him any difficulty, he will have dilated veins in the neck or ascites." He looked at the neck and there was no dilatation of the veins and there was no ascites. He then said: "He may have adhesive pericarditis, but if he has, it is not bothering him any. What's the next case?" I went back to my department much chagrined, but having learned a good lesson.

There is "adhesive" pericarditis. I believe that term should be retained, but adhesive pericarditis does not always mean compressive pericarditis. They are two distinct things, and we must differentiate them in terms of physiology, as Dr. Beck has so ably done this morning. It is abnormal physi-

ology, quite similar in fundamental concept to the excellent presentation of Dr. Golden last night.[1]

Dr. Beck asked me to take up the roentgen signs of this condition, but he has done it so well that I think anything more is unnecessary. If the heart is small, that is the most important thing. If the amplitude of the beat is small and if the scars are in the right place, you lose the a.v. differentiation, as we call it, that is, the visible difference in pulsation between auricle and ventricle, as you watch it. You see a difference in the time and amplitude of the beat. It is only later that the secondary signs of ascites, hydrothorax, etc., appear. The hydrothorax may obscure the heart and it may be only after the removal of the fluid that it is possible to see the small heart and diminished amplitude of the beat.

All these signs may fail in dry weather. The only sign that has not failed me in our series of cases is calcification in the pericardium. That has been reliable in 100 per cent of the cases, but even calcification in the pericardium does not mean compressive pericarditis or compressive extrinsic heart disease. That is one thing we must remember.

Finally, let me say that we all appreciate the excellent work which Dr. Beck is doing, and his thorough and illuminating presentation, both illustrating the value of concentration of effort.

[1] See Radiology **36:** 262, March 1941.

A. Carlton Ernstene, M.D. (Cleveland, Ohio): Chronic compression of the heart is a condition that can be recognized only with difficulty in its earlier stages, but should offer little trouble when the physical signs are well developed. Even in advanced cases, however, the true state of affairs often is overlooked, and this is to be regretted in view of the brilliant results which follow successful resection of the pericardial scar. As knowledge of these results is more widely disseminated, more interest undoubtedly will be aroused in the condition, and the diagnosis will be made more often. The most helpful diagnostic feature consists of the presence of congestion in the pulmonary and peripheral venous circulation in an individual with a diminished pulse pressure and little or no enlargement of the heart. The peripheral venous pressure is increased, the liver is enlarged, and, except in the earlier stages, ascites with or without edema of the legs is present. The contrast between extreme ascites and limited edema of the legs often is quite striking. Fluoroscopic examination and roentgen kymograms show diminished amplitude of the cardiac pulsations, and in a certain number of patients calcific deposits in the pericardium are readily demonstrable. Chronic compression of the heart is entirely a surgical problem. Medical management has nothing to offer other than such palliative measures as repeated abdominal paracenteses.

Roentgen Measurement of Visual Acuity in Cataractous Eyes[1]

R. R. NEWELL, M.D., and W. E. BORLEY, M.D.

San Francisco, Calif.

THE NATURE OF VISION BY ROENTGEN RAY

ROENTGEN (1) IN 1896 believed x-rays invisible, but in 1897 (2) reported that they could be seen by the unaided eye. Desjardins (3) as late as 1931 wrote that they are invisible. Taft (4) expressed astonishment that so easy a demonstration had been missed by almost all radiologists, and gave references to early workers whose writings in this respect had been ignored, among others Edison.

Since the appearance of Pirie's paper in 1934 (5, 6) we have used "Pirie's test" in cases of ocular foreign body, seeking to distinguish the single retinal shadow of an intraocular foreign body from the double shadow of an extraocular one, seldom with success.

Being curious about the unsettled question as to whether x-rays stimulate the retina directly or by production of fluorescence in some part of the eye, we irradiated in the dark the opened eyeball just removed from a rabbit after two hours' dark adaptation. The irradiation was about 200,000 times the threshold for x-ray vision, *i.e.*, at 100,000 r per minute (for one second). The lens fluoresced but the retina did not. Of course the brightness of the retinal image even with observers' pupils well dilated is only about 0.03 the brightness of the object viewed. That is to say, a fluorescent sheet would have to be 30 times as bright to see by looking at it, as it would need be if put in contact with the retina. There might, to be sure, be some fluorescent substance in the human retina that is lacking in the rabbit's retina. Indeed, we do not know whether a rabbit can see x-rays at all.

We thought it safe to irradiate a human eye at 1,000 r per min. (for 1 second three times). We chose a patient who had had the lens extracted together with its capsule. On observing her eye during irradiation at 1,000 r per min., from the side, no glow could be seen in the pupil. The lens of a normal eye fluoresced visibly when so irradiated, as did the skin, also. Patient and observers were, of course, completely dark-adapted.

We have calculated the amount of energy needed for vision by x-ray. The threshold value of 1/2 r per minute to a spot measuring 1 sq. mm. on the retina amounts to an absorption of 2×10^{-5} ergs per second in the 50 μ thickness of the rod layer. The threshold for light (7) is about 4×10^{-9} ergs per second. On an energy basis, therefore, light is 5,000 times as efficient as x-rays for retinal stimulation.

TABLE I

A	I		Product A × I	
Total Area of Double Dot (sq. mm.)	Threshold r per Minute			
	Observer B[1]	Observer M[2]	for B	for M
0.025	30.0	53.0	0.75	1.3
0.096	7.0	..	0.67	...
0.20	..	7.0	..	1.4
0.60	1.6	2.0	0.96	1.2

[1] B is a younger man and has a lower threshold than R. R. N., whose dark-adaptation curve is shown in Figure 1.

[2] M has believed himself somewhat deficient in vitamin A.

The best evidence that x-ray vision and ordinary vision are similarly dependent on visual purple is the similar course of dark-adaptation in both. Figure 1-*B* is a rough curve of dark-adaptation for one of us (R. R. N.).

X-ray and light both show a relative central scotoma for low intensities. We attribute this to the greater sensitivity of the rods compared to the cones, for the fovea contains only cones.

[1] From the Departments of Radiology and Ophthalmology, Stanford University School of Medicine, San Francisco, Calif. Presented before the Radiological Society of North America, at the Twenty-sixth Annual Meeting, at Cleveland, Ohio, Dec. 2–6, 1940.

X-ray and light both follow Ricco's law; *i.e.*, if one makes the size of a pattern twice as large (four times the area), it can be seen with one-fourth the illumination. This holds for light up to a 6-degree image (1.7 mm. on the retina). We have tested it for x-ray from 0.3 to 1.7-mm. patterns.

Within reasonable limits, therefore, one need pay little attention to the magnification and fuzziness of the roentgen pattern on the retina, but record only the size of the pattern in the stencil held against the eyelids.

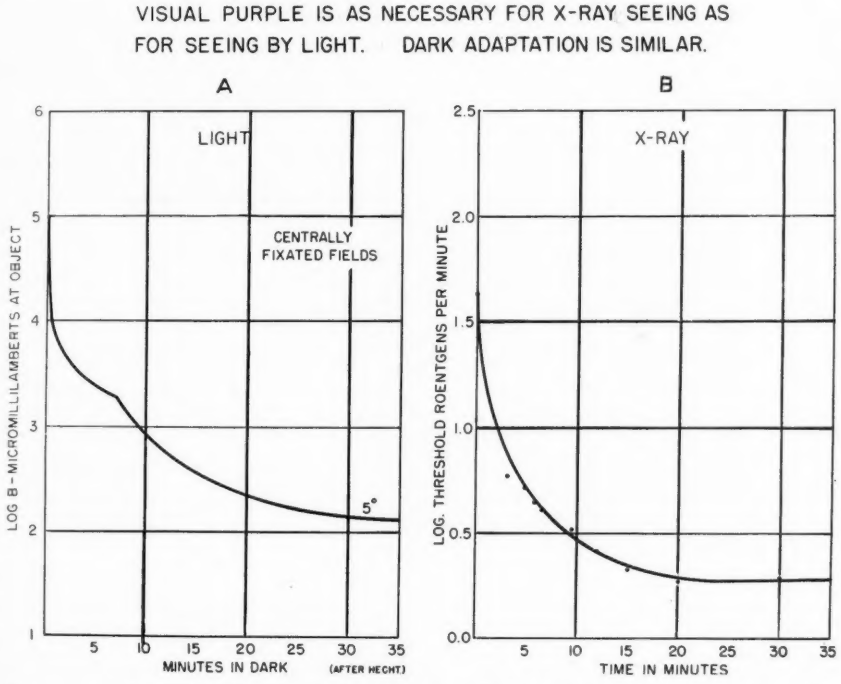

Fig. 1. *A*. Dark-adaptation curve for light. *B*. Dark-adaptation curve for x-ray. Note that log. scales are not the same.

MEASUREMENT OF VISUAL ACUITY BY ROENTGEN RAY

Because x-rays penetrate the media whether they are clear or not, it should be possible by use of them to measure visual acuity even when lens or cornea is opaque. Gifford and Barth (8) had some success in this. They used a stencil with simple geometric figures. Their smallest pattern was a single dot 0.25 mm. in diameter.

With light, vision is ordinarily tested by the reading of letters of various sizes. Normally in good light it is possible to read test letters subtending a visual angle of five minutes, with lines of a thickness of one minute. Such a letter forms a retinal image 25 microns across. Being unable to focus x-rays, one can only use a stencil to throw the x-ray spots on the retina. For small spots, this makes letters inconveniently complicated. We adopted, therefore, the simplest test form we could devise, namely, two round dots. We made them of equal size and set them 3 r apart (centers).

The smallest pattern we have yet made has holes 0.12 and 0.13 mm. in diameter and 4 r apart instead of the intended 3 r. Our best stencil was punched with fine sewing needles in a 0.25-mm. gold sheet. Figure 2 shows the complete stencil and

its roentgen silhouette. Figure 3 shows the size required at 14-inch reading distance to get the same pattern on the retina in light; the inset shows Snellen test type for visual acuity "20/200" at the same reading distance, but it must be borne in mind that these are to be read under good illumination.

To see this smallest pattern and recognize it as a double dot requires an x-ray intensity of about 30 r per minute. For easy seeing one ought to use 100 r per minute, which is difficult to attain except with a large focal spot or close distance. Either way one fails to get a sharp shadow.

tance to the retina is 25 mm. For an unsharpness of 0.125 mm. (the diameter of the hole in the smallest pattern) one must, therefore, put the stencil at 30 cm. from a 1.5-mm. focal spot. With a G. E. mobile unit at 76 kv. and 15 ma. we got 100 r per minute. The tube is rated to carry this for a few seconds. For the larger spots a smaller current can be used.

We have reached here a convenient limit to the test, an x-ray pattern on the retina corresponding in visual angle to what we call "15/200" on the Snellen test charts. If we can make smaller holes closer together in a stencil thick enough to give

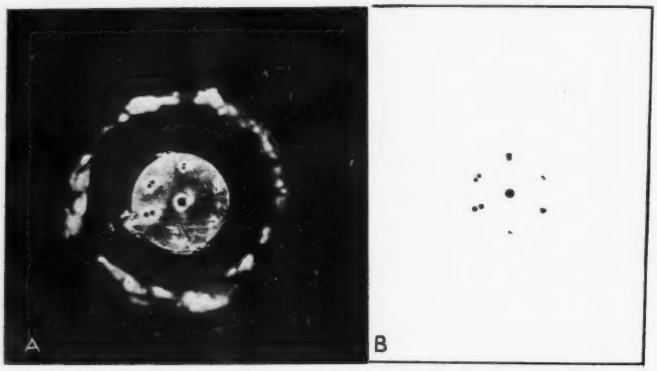

Fig. 2. *A*. Photograph of stencil (perforated gold plate) for x-ray test of vision. *B*. Roentgenogram of stencil.

To test the importance of fuzziness, we made a roentgenogram of the stencil with 1.5-mm. focal spot at 10.5 cm. and film at 2.5 cm. This gave an unsharpness of 0.36 mm. For the small holes this amounted to three times the hole diameter, but for the largest ones only about one-half the diameter.

Projecting a print of this onto a black screen with a Ferree-Rand projector, and sitting close or far, one could get a retinal image of almost any chosen size for any chosen degree of fuzziness. The test was done dark-adapted and with threshold illumination. Unsharpness of less than the diameter of the dot interfered but little with recognition of the orientation of a pair of dots.

With stencil against the eyelid, the dis-

reasonable roentgen protection, then it would be worth while using a rotating anode tube. Such tubes are available with focal spot 0.4 mm. in diameter. One might hope, therefore, to use holes of 0.04-mm. diameter (a tenth of the area we have so far attained), which would require ten times the x-ray for adequate stimulation, an output barely available from the rotating anode tube. This might discover impaired acuity no worse than "20/100." But intensity cannot be got high enough really to test central vision as the ophthalmologist knows it.

SAFETY

Filtered x-ray will, of course, be safer than unfiltered. Any shockproof tube will have inherent filtration to give a half

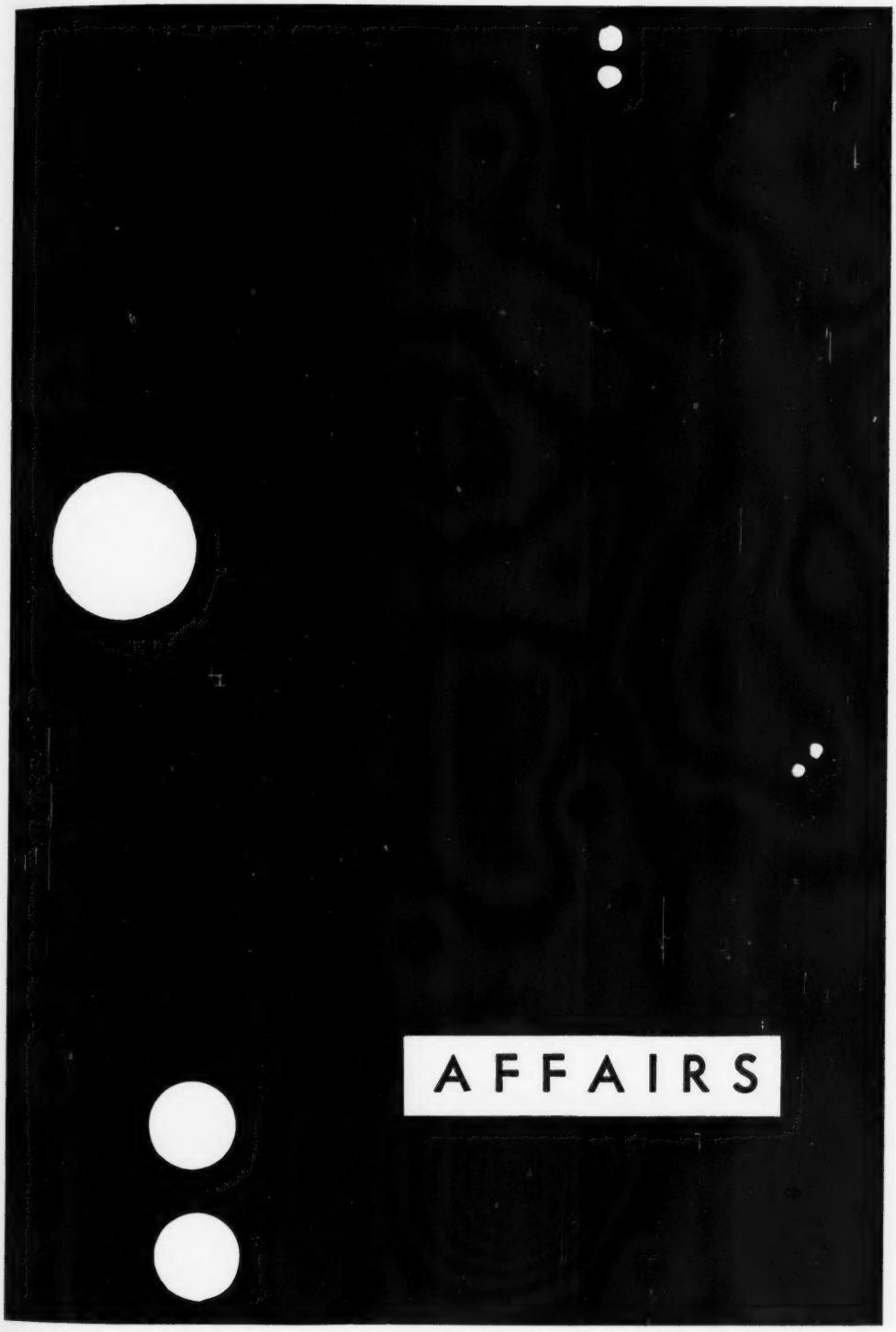

Fig. 3. At 14-inch reading distance, this gives a retinal pattern equal to that of the x-ray stencil shown in Figure 2. Inset are letters equivalent to Snellen "20/200" for 14-inch reading distance.

value layer equal to 1.5 or 2.0 mm. Al, giving retinal intensity about a third of skin intensity. To get a 50 per cen improvement would be easy, but to double the penetration one would have to go to 220 kv. and Thoraeus filter, which is quite out of the question.

Many published warnings can be found as to the sensitivity of the eye to roentgen injury. One erythema dose has been credited with production of cataract. Nevertheless, we have dared to give 500 r to the cornea for keratitis, and in a few cases of malignant tumor of the eye have given 2,000 to 3,000 r to half a square centimeter in one sitting, without disaster. Reese and Martin (9), in treating some 40 cases of retinoblastoma with very large doses of x-ray, have seen a few cases of cataract and phthisis bulbae follow.

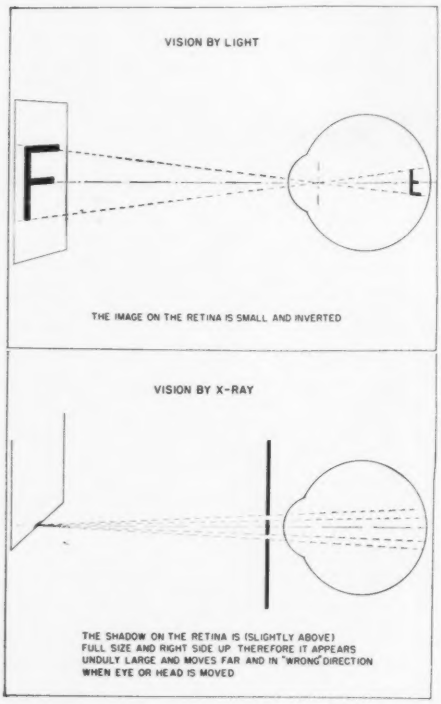

Fig. 4. Comparison of vision by x-ray and vision by light.

We have always started our test at 10 r per minute, adequate for seeing the larger patterns. We have often worked with a patient for an hour. If the exposure had been all in one spot, the total would have been 600 r, but the patient kept moving slightly in front of the stencil, so no one spot of skin or lens got more than a fraction of this total. Moreover, the area irradiated is very tiny. Safety standards for x-ray dosage are based on areas 10,000 times as large. On the whole, we believe there is no danger in an hour's use of x-ray for testing vision.

After the patient has learned the technic of seeing by x-ray, then for the smallest spots we use 100 r per minute, but for only two seconds at a time, perhaps half a dozen times (a total of 20 r).

CLINICAL APPLICATION

For a person untrained in optical science, x-ray seeing may be astonishingly difficult. The large size and small brightness of the spots is unanticipated from the appearance of the stencil (Fig. 4). The large movement of the spots for small movements of the head is disturbing. Moreover, when one does move his head toward the spots seen in peripheral field, they do not come into the central field, but jump in the opposite direction and are lost. Instinctive eye movements for fixation are, of course, ineffective.

An intelligent layman can learn to manage the test and bring any chosen dot into central (or paracentral) field of vision after a few minutes' experience. The clinical use of the test, however, is mostly in cataract cases, usually in elderly patients. It is discouraging to struggle with the shortcomings of an elderly intellect, which has lost almost all its youthful ability to cope with unfamiliar situations. We have often spent half an hour and occasionally a full hour on the test, which on medical students can be run off in a couple of minutes. We habitually measure each eye, starting with the one having the better vision. The test on the second eye usually goes quickly, the patient by that time being trained.

NORMAL EYES

The x-ray threshold of acuity for a score of normal persons proved not to be quite uniform. A few were found to be suffering from mild night-blindness, but we are not sure that night-blindness explained all the cases of poor x-ray vision.

SCOTOMA

The roentgen test in cases of central scotomata with clear media ought to give information as to its usefulness. We studied 24 such patients, varying in visual acuity from 20/40 to hand movements. Their defects varied from 2- to 3-degree relative scotomata to 20-degree absolute scotomata. The causes ranged from choroidoretinitis and macular degeneration to retrobulbar neuritis and toxic amblyopia.

The defect was usually detected by the x-ray test. In 17 of the 24 scotomata, x-ray pattern smaller than 10 degrees was not resolved (not seen as two dots). Three patients could resolve the 7-degree and four the 3 1/2-degree pattern. At the time these tests were completed we had no stencil with a pattern smaller than 3 1/2 degrees in total diameter. One of the four showing good acuity by x-ray became aware of the fact that she was using the field of vision peripheral to her blind spot. We have not yet worked out a satisfactory method of holding fixation during the test, although such would seem not impossible.

Our smallest present pattern, 0.36 mm. total diameter, subtends a visual angle of 1 1/4 degrees. Very few scotomata are as small as that. Given an intelligent patient, one ought to be able to detect a scotoma, a retinal detachment, or other gross abnormality that would seriously impair vision after the removal of a cataract or the inlaying of a clear window in the cornea (whichever kind of opacity is causing the poor vision).

Two cases were of especial interest. Both patients could distinguish and resolve the 3 1/2-degree pattern. One of them had follicular lymphoblastoma from which he later died. He had recently developed central scotomata. The fundi appeared normal. We could not detect the scotoma by x-ray. After x-ray treatment to the orbits the scotomata cleared and vision returned to normal. In the other case a central scotoma developed following a severe iridocyclitis and there was a questionable small cyst or hole in the macula apparently at fixation point. This patient went on to almost complete recovery after a period of one year, with a final visual acuity of 20/20. Is it possible that there may be some of these so-called "retrobulbar neuritis" cases with retinal infiltrate thick enough to stop a certain proportion of light rays, yet not visible with the ophthalmoscope, nor of sufficient destructive effect on rods and cones to render them unresponsive to x-ray stimulation?

CATARACTS

In this group there were 61 eyes with cataractous lenses. All of these cases were followed through operation and final visual acuity was determined with the best possible correction for aphakia. The majority of these cases were senile cataracts; a few were complicated by trauma and infection (uveitis); ten were associated with diabetes. As will be shown, there was a fairly close correlation between the performance of the test and the final visual acuity, and this in spite of the aforementioned difficulties of senility and strangeness of the procedure.

Of the 61 eyes after removal of the cataract, 43 had final vision of 20/30 or better. Thirty-four of these had given a good x-ray test, most of them resolving the 3 1/2-degree pattern (the smallest we had at that time). It is seen from this that good final vision was predicted successfully in 80 per cent of the cases.

There were 18 eyes with final vision of 20/40 or worse. Half of these had been unable to resolve even the 10-degree pattern by x-ray. In 50 per cent of this group, final vision was successfully predicted.

There are two groups of bad prediction to consider: (*a*) Nine eyes could resolve 7 1/2 or even 3 1/2-degree patterns by

x-ray, but ended with vision of 20/40 or worse. Such cases are unavoidable as long as we have to work with such coarse x-ray spots. Future tests will be carried down to 1 1/4 degree patterns. (*b*) Nine eyes with final vision of 20/30 or better had been unable by x-ray to resolve any pattern smaller than 10 degrees. A likely cause for such a discrepancy would be poor dark-adaptation. Especially suggestive is the fact that of 10 cases of cataract in diabetics, 4 fall in this group of good final vision with poor x-ray performance. Poor dark-adaptation is frequent in diabetes. McDonald and Adler (10) have shown that the course of dark-adaptation can be measured with the Hecht adaptometer even in presence of cataract. Although the threshold values are all higher, the cone-rod transition time and the form of the curve are normal. Suggested for the future is the study of the vitamin A content of the blood in those cataract patients showing poor performance of the roentgen test. If this is found to be low, efficiency in the test may be improved after administration of large quantities of vitamin A concentrate in some form. It would furthermore be of interest to check the dark-adaptation of those who after operation show unpredicted good vision.

We have applied the test in three cases of corneal opacity requiring transplantation. In each instance the 3 1/2-degree pattern could be resolved. Following successful keratoplasty in these cases, visual acuities were 20/20, 20/30, and 20/40, respectively.

SUMMARY

Physiology of vision by x-ray is reviewed as far as known.

In eyes with varying grades of diminished vision resulting from opacities of the media, the retinal function can be tested by the roentgen ray, irrespective of the type or density of the opacity.

Experience in 61 eyes before operation for cataract is encouraging. With attainable refinements of the test and attention to vitamin A nutrition, it would appear possible to avoid most cases of disappointment due to poor final vision after cataract extraction.

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DISCUSSION

A. B. Bruner, M.D. (Cleveland, Ohio): I have always been pleased to assume that the radiologist is the nearest to a combined laboratory man and clinician of any of the medical specialists. That probably explains why he is so open-minded to the clinician's point of view and so tolerant of the clinician's criticism. I shall discuss the paper which has just been presented from the standpoint of the clinician, and I can see a good many practical clinical objections.

The question of fixation was mentioned. The fact does not seem to have been taken into account that a very fair percentage of patients with cataracts have naturally poor fixation. This depends not only on the patient's intelligence quotient but also on the fact that fifty per cent of cataractous eyes are non-dominant eyes to begin with. We cannot overlook the sensitivity of the eye to roentgen damage, but that, naturally, Dr. Newell can discuss much more authoritatively than I can. In addition the time consumed seems to be too great for the results obtained.

Undoubtedly this test will give considerable information regarding peripheral function, but as regards macular function it is open to some question. The presence of central scotoma and retinal detachment is determined just as easily or more easily by simpler clinical methods.

As far as paracentral scotoma is concerned, it

can be overlooked at times by any known method. With this test the percentage of satisfactory results is no greater than with the ordinary clinical test. Another field wherein we might hope for help from such studies as these, but in which they apparently play no part, is the diagnosis of possible vitreous opacities before operation.

Finally I should think that any test presupposed to guarantee a successful result in cataract surgery is open to question, if for no other reason than that too many things can happen after the diagnosis is made. Good surgical judgment and technic will overcome a great many of the difficulties, but the individual skill of the surgeon and the co-operation of the patient are still primarily important to a good result.

R. R. Newell, M.D. (*closing*): The problem of fixation we have not yet solved. It may yet be that we can do roentgen campimetry by finding some way to achieve roentgen fixation. Without fixation, it is even more important that one should take the acuity measuring pattern down to an absolute minimum size. But ordinary clinical measurement of visual acuity (by Snellen test cards) does not require "fixation."

X-ray measurement of acuity is indeed time-consuming. It is not too time-consuming, however, if it gives the patient something which he cannot get in any other way.

I think Dr. Bruner misunderstood the purpose of our communication. Our intention was to discover a scotoma or a retinal detachment when opaque media make the diagnosis otherwise impossible.

I have been informed that the routine test that is given before removal of a cataract is light perception and light projection, the roughest possible test. No ophthalmologist certainly would dare remove a cataract without doing those tests to show that there is a functioning retina. If we can discover more about the function of the retina before removing the cataract, it seems to me we have done the patient much more good. If we cannot give a guarantee of good vision after removal of the cataract—that is true, we can't—we cannot discover vitreous opacity, which may still keep the patient from having good vision after removal of the opaque lens. We can, however, discover a certain number of patients to whom we can say: "It looks very doubtful that you are going to have an eye with which you can read after we remove your cataract. Do you still want to take the chance?"

There is one thing that we have found in eyes with clear media which may be a further help to the ophthalmologist. That is, we have discovered a few cases of scotoma in which the scotoma was there for light but the patient had normal x-ray vision. Such scotomas proved to be evanescent. One was a little cyst of some sort in the retina. The other was an infiltrate in a case of lymphoblastoma which cleared up under roentgen treatment.

I should like to add one more thing: The General Electric Corporation most generously loaned me a shockproof mobile unit for my demonstration, and I wish to thank them for their courtesy.

Melorheostosis, with Report of a Case[1]

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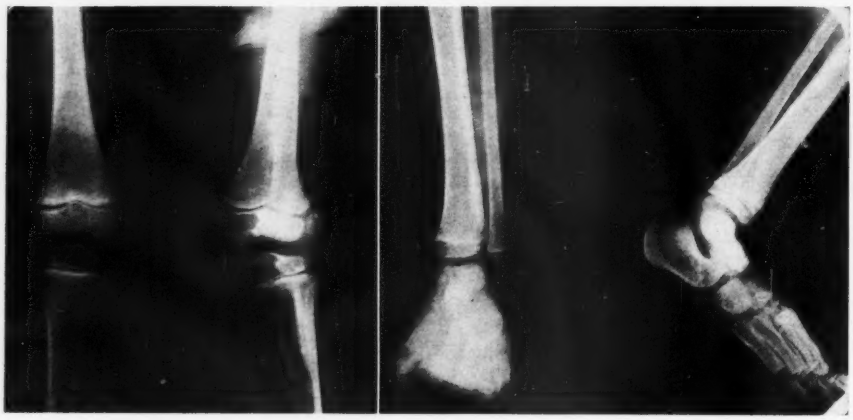
MELORHEOSTOSIS was first described by Léri and Joanny, in 1922. The term is derived from the Greek words μέλος, a member, and ῥεω, to flow. Léri described the condition as "a flowing hyperostosis of a single extremity," and reported two cases. He pointed out that it was a hitherto undescribed type of bone hyperplasia confined to an extremity, extending from one end to the other in a single linear tract, but usually leaving a few unaffected areas along the course.



Figs. 1 and 2. Melorheostosis in a child of five: roentgenograms made in 1931.

CASE REPORT

Alice I., a Japanese girl, born in this country, five years of age, was referred to the writer by Dr. Y. S. Fukuda, at the Japanese Hospital, Los Angeles, Nov. 2, 1931. She had difficulty in walking and some valgus deformity of the left knee. There had been slight pain in the left leg at times. The onset had been gradual. The mother stated that the child had been late in starting to walk and had had a slight limp. Physical examination was negative except for moderate genu valgum. Routine blood and urine tests and blood Wassermann tests were negative. There was no significant family history. There was no muscular atrophy and no shortening of the bones of the left leg. The limp appeared to be due to the valgus deformity. No biopsy was obtained.

Roentgenograms of the left knee, left hip, pelvis, and left ankle showed linear areas of increased density extending from the head of the left femur along the medial and anterior surface of the shaft, ending in a streak about a quarter of an inch above the distal epiphysis. The epiphysis of the femur at the knee showed an area of increased density involving the central portion of the anterior and of the articular surface. In the patella, which was displaced laterally, there was a spotty condensation of bone. The upper epiphysis of the tibia also showed a spotty condensation. Osseous tissue about the areas of condensation was decalcified. The tibia was rotated outward and there was a subluxation at the knee joint. A streak of condensation extended down the lateral and anterior surface of the tibia to the lower epiphysis. The fibula was not involved. A large area of condensation was seen in the astragalus, and there were traces in the os calcis, navicular, and first metatarsal. There appeared to be arrested development of the epiphyses at the involved sites, and normal growth of uninvolved bone.

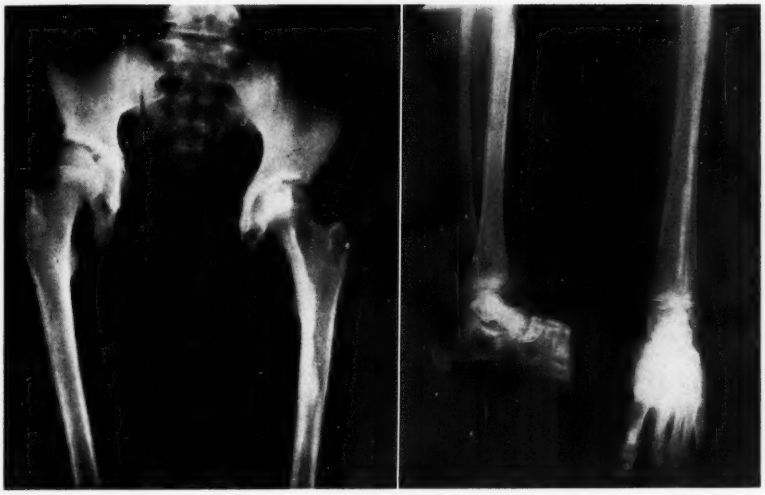
Recognizing the unusual character of the lesions, the writer sent the roentgenogram and history to Dr. J. C. Bloodgood, who commented as follows: "There is no doubt from an x-ray standpoint it is an area of sclerosed bone, and, in view of the nondevelopment of the epiphysis of the tibia and of the astragalus, it must be an old affair—perhaps intrauterine. I know no literature on it. Diagnosis:

[1] Accepted for publication in February 1939.

Multiple sclerosing osteitis or osteomyelitis—lower epiphysis of the femur—upper epiphysis of the tibia—shaft of the tibia—astragalus—with genu valgum."

Dr. C. F. Geschickter, who also saw the roentgenograms, wrote as follows: "The x-rays show increased bone density extending down from the femur through the knee joint into the astragalus on one side only, suggesting melorheostosis. We have a case..... reported in RADIOLOGY, February 1931."

In November 1938 the writer requested the patient to return for further roentgenographic studies, to see what changes had taken place in the condition in the interval since the previous roentgen studies. The child had meantime been under the care of Dr. Charles Young, of the Orthopedic Hospital, Los Angeles, who had operated on her five and four years previously to correct the deformities of the lower end of the left femur, the position of the patella, and the alignment of the tibia at the knee joint. He reported encountering very dense bone, but no pathologic examination was done.



Figs. 3 and 4. Roentgenograms made in 1938 of patient shown in Figs. 1 and 2, now eleven years old.

The roentgenograms showed no increase in the extent of the lesions or any alteration in their distribution. The bones of the left leg were slightly smaller than those of the right, and, except for the areas of condensation, appeared to contain less lime salts. Growth at the epiphyses did not appear to have been appreciably retarded. The left leg showed one inch of shortening, which was partially compensated for by a corrective shoe. The left leg was smaller than the right, and the patient walked with a slight limp, but was as active as a normal child of her age.

REVIEW OF LITERATURE

Three years after the appearance of the report by Léri and Joanny, Lewin and MacLeod published case. They stated that their roentgenograms had been shown to some of the leading roentgenologists of the United States and Canada, to several general surgeons, to some international authorities on bone pathology, and to several neurologists, and that not one had ever seen or heard of a similar example. Their patient was a male, thirty-five years old, with peculiar swellings of two fingers of the right hand, first noticed at the age of six, and dull, constant pain since the age of ten, growing progressively worse. Roentgenograms showed "eburnation and sclerosis with almost complete disappearance of the medulla, due to encroachment on the canal by the hyperostosis." The lateral aspect of the ulna, triquetrum, pisiform, capitatum, fourth and fifth metacarpals, and the phalanges of the fourth and fifth digits was involved. The involvement, as the authors state, suggests the distribution of the ulna nerve.

Muzii, in 1926, reported a case in a girl, ten years of age, who had had symptoms since she was five years old. There was

involvement of the left lower limb from the superior pubic ramus, upper rim of the acetabulum, head and medial aspect of the femur, patella, tibia, talus, and all of the lesser bones of the medial half of the foot to the first digit. Coincidentally, there was craniostenosis.

In 1927, Putti reported the fourth case. The patient was a girl eight years of age, whose mother had noticed a pes valgus when the child was eleven months old. Pain began when she was seven. There was shortening of the left leg with slight bowing and genu valgum. The lip of the acetabulum, the shaft, great trochanter, lateral condyle of the femur, lateral aspect

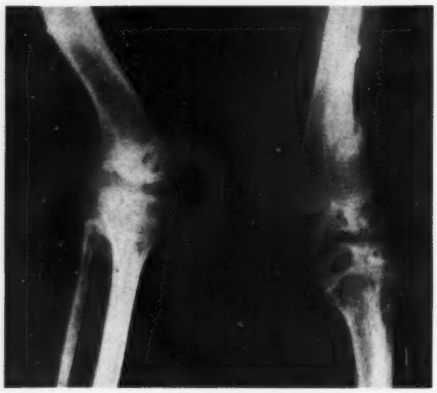


Fig. 5. Roentgenogram made in 1938 of patient shown in Figs. 1 and 2, now eleven years old.

of the tibia, astragalus, a portion of the os calcis, and the third cuneiform were involved. Biopsy showed an increased vascular pattern, obliteration of the vessel lumina, perivascular ossifications and atrophic marrow, few osteoblasts and many osteoclasts. Putti believed the condition to be due to local sympathetic contractions which preceded the osteogenic changes. Policard, of Lyons, studying the same tissue, considered the degenerative process primary and the condensations secondary, with the vascular obliterations coincidental. No etiologic factor was found by either investigator.

Cases were reported by Zimmer, in Germany, and by Perussia and Meda, in France, in the same year (1927). Zimmer's case was that of a man, thirty-two years of age, who had hyperostosis of the lip of the left acetabulum, the entire femur, including the head, neck, and lateral aspect of the shaft, and the lateral condyle. Skipping the knee joint, the process involved the fibula, talus, calcaneus, third cuneiform, and the third and fourth metatarsals. Zimmer considered the distribution of the densities to be the result of embryonic metameric disturbance. Biopsy specimens from this case, examined by Kaufmann, were reported to show compact trabeculations, normal haversian canals, and fibrosis of the bone marrow containing islands of osteoblasts.

In 1928, Meisels reported the eighth case, that of a woman, twenty-five years old, with involvement of the right lower extremity sparing the tibia. Meisels thought that when the epiphyseal-metaphyseal junction was involved previous to the completion of ossification, diminished growth resulted, but that excessive condensation and over-production of osseous tissue occurred after the growth stage had been completed, resulting in locking of the joints and bowing of the long bones.

Kemkes, in Germany (1929), had the oldest patient on record. A fifty-four-year-old machinist complained of dull intermittent pain and slight wasting of the upper right extremity, which he had noticed since the age of forty-two. In this case the right scapula, lateral aspect of the humerus, radius, lateral row of carpal bones, and the phalanges of the first and second digits were involved.

In 1930, Léri and Lièvre published a six-year study of Léri's original case. They described the development of new osseous condensations, proving roentgenologically the progressive character of the lesions. They tried to graft a section of the affected bone on the femur of a monkey to determine the infective or non-infective nature of the condition. The result was a complete absorption of the graft without production of similar changes.

In 1931, in Italy, Piergrossi presented a

case showing characteristic hyperostoses of the right ileum, ischium, and pubis, including the acetabulum. The lesions were less extensive along the lateral aspect of the femur. The tibia was not involved, but the fibula showed heavy hyperostosis and bowing. The lateral row of tarsal bones, metatarsals, and phalanges were also involved. The hip and knee joints showed narrowing and limitation of motion but were not fused. Piergrossi did not favor the developmental defect theory of Zimmer, but, rather, the local ischemia idea of Putti. His view was that a periostosis developed upon a primary simple osteosclerosis.

Weil and Weismann-Netter described a case in 1932. The patient was an army officer, thirty-seven years old, with involvement of the right upper extremity and of the entire right fourth rib. The radius and ulna were uninvolved, although the humerus and a few of the carpal bones were affected. These writers advocated the use of the term "*rhéostéose.*" Other commentators have pointed out that an involved limb and contiguous bony structures would be developed embryologically from the same anlage.

In 1932, Kraft listed 16 cases which had been reported in the literature in the past ten years in the following countries: Italy, 5 cases; Germany, 4; United States, 3; France, 2; Poland, 1; Sweden, 1. The ages ranged from eight to fifty-four years; the age at onset from five to forty-two years. Six patients were females and 10 were males. The upper left extremity was involved in 2 cases; the upper right in 6; left lower in 5, and right lower in 3.

Moore and De Lorimier also reviewed the literature, up to 1933, and added one case. These authors noted as characteristics an osteitis, as mentioned previously by Léri in the left patella, epiphysis of the left femur, and tibia, and as observed in the case here reported, in the astragalus. Moore and De Lorimier suggest that subperiosteal telangiectases might account for the pattern of distribution, which could be of congenital or developmental origin (as in purpura telangiectoides), due to toxins or trauma, or in response to allergic or endocrine conditions. This, these authors think, would explain the diverse biopsy findings. The localization of the lesions and distribution along one extremity, however, would seem to indicate that an endocrine factor could be but contributory. Moore and De Lorimier also suggest that the "progressive muscular weakness" noted in some of the cases may be due to eburnation of the bone about the insertions and origins of the muscles.

In 1934, Aldenhofen, reported the case of a forty-nine-year-old locksmith, formerly a seaman, who had lived a short time in South America. The patient's complaint was "lumbago," first noticed in the fall of 1917, and lasting for about six months but responding to applications of camphor liniment. Since 1932, he had had back pain, which gradually increased in severity. At times bending was impossible; more recently walking had become difficult, as well as rising from a sitting posture, and household remedies had failed to give relief. Examination revealed tenderness over the dorsal and lumbar vertebrae and sacrum, but no pain on motion of the spine. There was no noticeable limitation of active or passive movements of the extremities. Stooping caused cramp-like pains in the leg muscles. Examination of the nervous system and of the blood and urine revealed nothing of significance. Spinal and blood Wassermann tests were negative.

In the roentgenogram the left half of the pelvis appeared of greater density than the right. The condensation stopped at the sacro-iliac articulation and there was no trace of it in the spine. The symphysis pubis formed a sharp boundary below. The sclerosing process was most dense in the pubic bone. The condensation was symmetrical and general. Bone detail could not be seen. The femur showed the process but less extensively. The inner side of the head and shaft showed increased cortical density, as compared with the other side. In no other part of the skeleton

was any similar change found, nor was there any change in normal bone contour.

Aldenhofen points out that this condition differs from a somewhat similar case of osteitis condensans of the ilium, described by Bársony and Polgár, in several respects. The latter condition is frequently bilateral, involves the sacro-iliac articulation, and does not spread from the pelvis. The condensation is ring-shaped and there is no abrupt passing from normal to abnormal as in melorheostosis. There was no evidence in Aldenhofen's case of local trauma or an inflammatory process either of bone or adjacent tissue, as described by Clairmont and Schinz in osteitis condensans. Tuberculous infection, tumor, and syphilis were ruled out. The author would not hazard a conjecture as to prognosis. His studies furnished no clue as to etiology. He did not consider a possible head injury, malaria, or hereditary factors as in Albers-Schönberg's generalized osteosclerosis to be present in this case. The rheumatic complaint was, he considered, the factor which led to the accidental discovery of the bone condition.

In 1936, Dillehunt and Chuinard reported the case of a boy, ten years of age, who had had symptoms since he was six months old. The left leg was affected in the lateral aspect only; the knee was stiff, and the leg was one inch shorter than the right. The child was admitted to the hospital after having been struck on the left thigh with a baseball bat, but there was no history of previous injury. Physical examination revealed limitation of motion of the hip joint in all directions and a thickening of the skin and subcutaneous tissue from the anterior superior spine of the ilium to the foot. There was no pain on joint movement and no muscular atrophy. Routine blood and urine examinations were negative, and blood calcium was normal. There was no definite symptomatology suggestive of a clinical syndrome. No treatment was given and no biopsy taken.

Dillehunt and Chuinard summarize briefly the findings in the 19 cases which had been published previous to their report. Only four cases had been reported as originating after the bone growth period. The youngest patient was a five-year-old child. Onset is insidious and progress slow. Symptoms are more pronounced in children; radiologic changes more marked in adults. Weakness and wasting are usually to be found in the affected limb. Bowing is frequent but not in proportion to the extent of the lesion. Shortening or enlargement of the affected limb is rare, but there may be a slight limp. There is occasional stiffness of joints, some limitation of motion, rarely ankylosis. Induration or erythematous areas of skin may be present. Fractures have never been observed in the affected bones. Five biopsies have been reported, but there has been no characteristic appearance, and findings have not been in complete agreement. Some cases show an enlarged vascular pattern, some obliteration of vessel lumina, perivascular ossification, atrophic bone marrow rich in osteoblasts, compact lamellae, and islands of cartilage.

The diagnosis has been by roentgenographic evidence alone. Treatment has been symptomatic; heat, massage, and mild analgesics have been used. X-ray therapy has been recommended because some cases have seemed to improve after repeated exposures. There is no instance of surgical treatment and no report of autopsy findings. Malignancy, tuberculosis, and syphilis seem to play no part in the cases reported. Usually the joints are spared.

A case was reported from Norway in 1936, by Natvig. The patient, a forty-year-old man, had lesions on the right side involving the femur, patella, tibia, talus, navicular, first and second cuneiform, second metatarsal, and the first phalanx of the great toe. The fibula was spared, as in Putti's case and the one reported in this paper, but there was involvement of the superior ramus of the pubis and a portion of the acetabulum. These regions were not involved in Putti's patient or in the writer's case. The involved epiphyses

showed a more patchy distribution of areas of condensation than the diaphyses. There were shortening of the involved leg and bowing of the tibia, or weight-bearing bone, which seems to have characterized most of the adult cases involving the lower extremity. Upon questioning, after discovery of the bone condition by roentgen examination, the patient gave a history indicating duration of symptoms for ten years. The diagnosis on admission was lumbago. The patient had had syphilis, and, as in one of the Italian cases, malaria. Natvig believed that neither was a factor in the production of the bone lesions, as symptoms were manifest prior to the development of disease. Having reviewed the microscopic findings of the earlier investigators and the various theories advanced as to etiology, he concluded that the theory of Zimmer, namely, a congenital developmental defect in a primitive segment, explains the distribution and early occurrence of most of the cases but hardly accounts for the disease in adults.

SUMMARY

The literature on melorheostosis has been reviewed and one case has been added. Although we have been able to contribute nothing new in the way of etiologic or biopsy studies, it is hoped that this report may stimulate other roentgenologists meeting this condition to further study.

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Diaphragmatic Hernia of the Stomach[1]

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DIAPHRAGMATIC hernia is a protrusion of the abdominal contents into the chest through an abnormal opening in the diaphragm which results from imperfect development, anatomical weakness, or trauma. Recent statistics show that diaphragmatic hernia due to trauma is relatively uncommon, comprising less than one-third of the reported cases. In the author's series of 26 cases, 4 were considered as acquired diaphragmatic hernias of the stomach. It was noted in this study, in agreement with the reports of others, that protrusion of a portion of the cardiac end of the stomach through the esophageal hiatus was the most common type of hernia. In the congenital type the defect is usually caused by a failure of fusion mainly in four parts of the embryonic diaphragm, and the herniated organs may lie free in the chest with or without a peritoneal sac. A hernia may occur after fusion has taken place when a subsequent increase in abdominal pressure causes a protrusion through some weak area.

CLASSIFICATION

Numerous classifications have been presented based on embryology, anatomy, etiology, etc., but since it is often impossible to distinguish the various classes clinically, it is better, for academic reasons, to consider hernias as of two types, congenital and acquired.

The congenital group may be subdivided into (*a*) the form in which there is a complete absence of the diaphragm or a portion of the diaphragm; (*b*) the type which is due to a partial defect in the musculature of one leaf; (*c*) the form which is due to a congenital enlargement of one or more of the diaphragmatic hiatuses. The acquired type may be considered under the following subdivisions: (*a*) the form which is produced by a dilatation or relaxation of the various hiatuses of the diaphragm; (*b*) the form which is a result of trauma.

Not to be confused with the above conditions are (1) the thoracic stomach associated with a short esophagus, as in this instance the stomach has never been a part of the abdominal cavity and, therefore, cannot have herniated, and (2) so-called eventration of the diaphragm (in which the stomach, as well as the rest of the abdominal contents, is below the diaphragm) except in the presence of a congenital diaphragmatic defect. The author has one such case in his series.

INCIDENCE OF DIAPHRAGMATIC HERNIA

This condition can no longer be considered as a pathological curiosity, since Hedblom, in 1931, was able to collect 1,003 cases reported in the literature after 1900. From 1900 to 1925 only 30 cases of diaphragmatic hernia were clinically recognized at the Mayo Clinic; from 1925 to 1933, 147 cases were recognized, five times as many in a period of eight years as in the previous twenty-four years.

Truesdale in 1935 collected 303 cases which occurred in infants and children; of this number, 165 were found at autopsy, 90 by x-ray examination, and 13 at operation.

Pancoast and Boles discovered 16 diaphragmatic hernias among 9,000 gastric cases examined; all but one were nontraumatic. Carman in 1924 gave the ratio at the Mayo Clinic as one in 18,000 examinations, whereas in the author's series there were 26 in 7,000 cases. Either this condition is increasing in frequency or many cases have formerly been overlooked; it should be borne in mind, however, that

[1] From the Radiological Department, Augustana Hospital, Chicago. Presented before the Radiological Society of North America, at the Twenty-sixth Annual Meeting, Cleveland, Ohio, Dec. 2–6, 1940.

automobile accidents are producing an increased incidence of traumatic hernias.

EMBRYOLOGY

The pericardial and pleural cavities and the diaphragm are so closely related in their development that they must be considered together. In the region just caudal to the visceral arches where the two anlagen of the heart appear, the embryonic coelom becomes dilated at a very early stage to form the primitive pericardial cavity. This early pericardial cavity is simply the cephalic end of the embryonic coelom and is, therefore, directly continuous with the rest of the coelom.

The communication between the pericardial cavity and the rest of the embryonic coelom is soon partly cut off by the development of a transverse fold, the septum transversum. The partitions forming the septum transversum close the frontal part of the communication between the pericardial cavity and the rest of the coelom. The pleural cavities become separated from the pericardial cavity apparently through the agency of the duct of Cuvier, which extends from the body wall through the dorsal free edge of the septum transversum to join the sinus venosus. This free edge is pushed farther and farther into the ductus pleuro-pericardiacus until it meets and fuses with the mesentery or posterior mediastinum. This process thus produces a septum between each pleural cavity and the pericardial cavity. The septum transversum early acquires still more complicated relations from the fact that the liver grows into its caudal part. It may for this reason be divided into a caudal part, in which the liver is situated, and a cephalic part, which may be called the primary diaphragm. These two parts at first are not separated, the separation taking place secondarily.

After the separation between the pericardial cavity and the pleural cavities, the latter for a time remain in open communication with the rest of the coelom or peritoneal cavity. The lungs as they develop grow into the pleural cavity until their tips finally touch the cephalic surface of the liver. At this point, folds grow from the lateral and dorsal body walls and unite ventrally with the primary diaphragm and medially with the mesentery. These folds, the pleuro-peritoneal membranes, separate the pleural cavities from the peritoneal cavity and complete the diaphragm.

Thus the diaphragm, from the standpoint of development, consists of two parts, a frontal part, which is the cephalic portion of the original septum transversum, and a dorsal part, which develops later from the body wall and is the closing membrane between the peritoneal and pleural cavities. The principal changes which bring about adult conditions are the growth of the lungs, the separation of the diaphragm from the liver, and the caudal migration of the diaphragm itself. The diaphragm, during its development, migrates from a position in the cervical region, where the septum transversum first appears, to its final position opposite the last thoracic vertebra. This fact accounts for the innervation of the diaphragm by cervical spinal nerves. During the migration, the plane of direction also changes several times.

ANATOMY

The diaphragm is a single independent muscle; its shape differs from that of the skeletal muscles in that it is thin and dome-shaped, stretching across the inferior aperture of the thorax in such a way that it is convex toward the thorax and concave toward the abdomen. It consists of a central tendinous portion, the central tendon, and of a peripheral muscular portion.

The muscular fasciculi of the diaphragm are subdivided according to their origin into three parts, which are designated the sternal, costal, and lumbar portions. Of these, the lumbar portion is the strongest, and the sternal portion by far the weakest. Fibers of all three portions are inserted into the margins of the central tendon. The sternal portion arises from the posterior

surface of the xiphoid process and consists of but a few slender fasciculi.

The costal portion arises by broad fleshy serrations from the inner surface of the six lower costal cartilages, and from the eleventh and twelfth ribs, being also attached to the lumbar costal ligament in this situation, interdigitating with the transversus abdominalis and with the transversus thoracis following the curvature of the dome of the diaphragm and passing to the central tendon.

The greater part of the lumbar portion comes from the bodies of the lumbar vertebrae. Upon either side there may be distinguished three crura or pillars, the crus mediale, intermedium, and laterale. The inner tendinous margins unite at the level of the twelfth thoracic vertebra to form a pointed arch with tendinous margins which is converted into a short canal by the anterior surfaces of the last thoracic and first lumbar vertebrae. The opening so formed gives passage to the aorta and is consequently designated the aortic opening. The central tendon is a fibrous layer which may be either reniform or shaped like a clover-leaf, and its fasciculi undergo manifold decussations. The convex surface of the central tendon is situated anteriorly; the more marked concavity is placed posteriorly. In it there may be recognized a medial, slightly curved portion, which is situated between the two domes of the diaphragm, and two leaflets which are directly posterior. The left leaflet is the smaller and forms the left dome of the diaphragm; the right is larger and forms the right dome. At the base of the right leaflet near its posterior margin is situated a large, irregularly rounded opening completely within the central tendon, which gives passage to the inferior vena cava. The right dome is more capacious and extends to a higher level than the left.

The diaphragm possesses a series of foramina and spaces which give passage to vessels or nerves. These are: (1) the aortic opening, which is only partly formed by the diaphragm; (2) the esophageal opening, purely musculature and formed entirely by the diaphragm; (3) the opening for the vena cava, situated entirely within the tendinous portion of the muscle; (4) the slit-like spaces between the inner and middle crura and between the middle and external crura. The latter spaces give passage to the vena azygos, to the vena hemiazygos and to the sympathetic and splanchnic nerves. In addition to the aorta the aortic opening also transmits the thoracic duct. The motor nerve of the diaphragm is the phrenic from the cervical plexus and the fifth or sixth lower intercostal nerve. Small filaments are also sent to the diaphragm from the diaphragmatic plexus, which are derived from the semilunar ganglia and solar plexus.

SYMPTOMATOLOGY

The symptoms of diaphragmatic hernia, which are usually the result of mechanical interference with the functions of the herniated viscera and not infrequently due to interference with respiratory and circulatory organs upon which they encroach, may be respiratory, gastro-intestinal, or both, and are very protean in character.

An analysis of the symptoms in the author's 26 cases reveals the following. The cardinal symptom was pain, which was present in 22 cases. In over one-half of these the pain was at the epigastrium, in the remainder elsewhere in the abdomen. The next most frequent symptom was indigestion or gas or belching after eating, present in over one-half the cases. The patient not infrequently obtained relief by sitting up and forced vomiting. Vomiting occurred in 8 cases; 3 patients vomited blood. Dysphagia or choking spells occurred in one-fourth of the cases. The other symptoms were shortness of breath, weakness, and loss in weight, which ranged from 5 to 40 pounds in one year.

PHYSICAL EXAMINATION AND LABORATORY FINDINGS

The physical examination revealed nothing particularly remarkable except that

two patients were acutely ill, both of whom had acquired hernias.

The laboratory findings were likewise of little interest with the exception of secondary anemia in two cases, which was due to associated pathology.

DIAGNOSIS

A review of the literature, as well as an analysis of the cases reported by other clinicians, shows that the clinical diagnosis of diaphragmatic hernia of the stomach is difficult. The diagnosis is greatly facilitated and can usually be made preoperatively by a competent roentgenologic examination of the gastro-intestinal tract. In 6 of the 26 cases reviewed here the clinical diagnosis was a question of diaphragmatic hernia; in 6 a question of ulcer of the stomach or duodenum; in 3, a question of gallbladder disease; in 4, a question of carcinoma of the stomach; in 3, a question of cancer of the esophagus, and in the remaining 4, a question of chronic cholecystitis, myocardial disease, cardiospasm, and indeterminate.

A thorough fluoroscopic examination of the gastro-intestinal tract may reveal air or gas bubbles in the chest, an elevation of the diaphragm, irregularity of the diaphragm, and displacement of the lower end of the esophagus, usually with some dilatation and sometimes tortuosity. When the patient is examined in the recumbent position, either prone or supine, and in the Trendelenburg position, a portion of the stomach may be seen above the shadow of the diaphragm.

The roentgenologic diagnoses in the 26 cases were 22 congenital and 4 acquired hernias. Twenty-one of the congenital hernias were paraesophageal and one was through the tendinous portion of the left diaphragm. Of the acquired hernias, two were paraesophageal; one was through the posterior half of the left diaphragm at the outer three-fifths and one through the mid-portion of the left diaphragm, 3 inches from the esophagus and extending laterally for 6 inches. The hernias involved the cardiac portion in 22 cases and in 4 practically the entire stomach was in the chest and inverted, so-called "upside-down" stomach.

SIZE OF HERNIAS AND INVOLVEMENT OF THE DIAPHRAGM AND ESOPHAGUS

Fifteen of the hernias were the size of an orange; 8 the size of an apricot or peach, and 3 the size of a grapefruit.

In the congenital hernias there was no elevation of the diaphragm, no paralysis, no irregularity, no paradoxical excursion. In the acquired hernias, exclusive of the paraesophageal, the diaphragm was elevated, paralyzed, with paradoxical excursion, and irregular in outline. The findings at the lower end of the esophagus are important in determining the possibility of diaphragmatic hernia of the stomach. The lower end of the esophagus was displaced to the right in 17 cases, displaced posteriorly in 2, and to the left in a single case. Variant degrees of obstruction or cardiospasm were seen in 11 cases. The heart was displaced to the right in 3 patients.

ASSOCIATED PATHOLOGY

The associated pathology in this series was of some interest; one patient had a carcinoma of the stomach; three, duodenal ulcer; one, diverticulitis of the esophagus; one, diverticulum of the cardiac portion of the stomach, and one, cholecystic disease with cholelithiasis.

TREATMENT

The treatment of diaphragmatic hernia of the stomach in which the manifestations are mild and there is, therefore, no obstruction, may be medical, as many patients may go through life without severe enough symptoms to warrant operative interference. In acute cases due to indirect injury to the diaphragm without injury to the viscera, palliative measures are probably preferable until the shock of the injury and the acute symptoms subside. As pointed out by Harrington, palliative treatment may consist in passing a stomach tube and giving an enema to empty

the gastro-intestinal tract of gas, thus diminishing the size of the displaced viscera and permitting a return to their normal position. Hedblom believed that a small hernia anywhere except at the esophageal hiatus should be repaired even in the absence of symptoms. In any event if the symptoms get progressively worse, if there are signs of strangulation or obstruction, or any indications of an acute surgical condition, palliative interference should be instituted. Operative replacement of the herniated viscera in the abdomen with repair of the abnormal opening in the diaphragm is the only treatment that insures complete relief of symptoms. Many believe that the operative treatment should be carried out as soon as a diagnosis is made, particularly if the intestines are involved in the hernia, in which case it is usually of the traumatic type.

Preliminary phrenicotomy is often of value in the repair of enlarged hernial openings if there has been considerable loss of structure. Opinions as to the surgical approach differ. Some authors prefer the abdominal, some the thoracic, and others a combination of the two. Harrington prefers the abdominal approach. Hedblom believed that the thoracic and abdominal approach each had its special advantages and limitations depending upon the anatomic and clinical type of hernia. Donovan states that positive pressure anesthesia is decidedly advantageous; it was employed in most of his cases. He also believes that these patients should be kept in an oxygen tent for several days after operation. Not only does the oxygen decrease the respiratory effort, thereby saving the patient's energy, but the tent decreases the chances of respiratory infection because the temperature can be so easily regulated. Pleural effusion may follow operation; if it occurs, the chest should be aspirated as often as the quantity of fluid demands.

Of the 26 cases studied, 8 were surgical and 18 were treated symptomatically. Of the 8 patients operated upon 7 had an uneventful recovery and one died. One patient had a moderate recurrence of the hernia six months later. In the one case that came to autopsy, the entire stomach and splenic flexure were in the left pleural cavity. The tendinous portion of the left diaphragm was defective, with a rent 10 cm. long. The stomach and colon were in a sac of serous membrane. The patient died from shock.

CONCLUSIONS

The symptoms of diaphragmatic hernia of the stomach may be either respiratory, gastro-intestinal, or a combination of both, and in some instances so protean in character as to be indeterminate.

A small percentage of 26 cases here reviewed were relatively symptom-free and the hernia was discovered in the course of a routine roentgenologic examination.

The diagnosis of diaphragmatic hernia is most easily and completely made by a thorough roentgenologic examination of the gastro-intestinal tract.

Operative replacement of the herniated viscera into the abdomen with repair of the abnormal opening in the diaphragm is the only treatment that insures complete relief of symptoms and should always be done when the clinical symptoms warrant.

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DISCUSSION

George Crile, Jr., M.D. (Cleveland, Ohio): Dr. Beilin's paper on diaphragmatic hernia is particularly interesting and timely because of the increased frequency with which this condition is being recognized. The majority of our cases have been discovered in the past five years.

Unfortunately, perhaps, the clinician's understanding of hiatus hernia has failed to keep pace with the roentgenologist's increased diagnostic acumen. Too frequently the physician does not have a clear conception of the pathology of the lesion or of the technic of the operation required for its correction. He fails to differentiate between simple esophageal hiatus hernia, which is the most common type, constituting in the neighborhood of 80 to 90 per cent of all cases seen, and the extensive traumatic ruptures of the diaphragm with all of the viscera within the thoracic cavity.

The importance of making that differentiation lies in the fact that the treatment is so different. In the true traumatic type, in which the colon, stomach, spleen, and other abdominal organs are involved, an extensive operation, oftentimes involving both a transpleural approach with resection of ribs and an abdominal approach as well, is usually indicated. This carries with it, naturally, a much higher mortality rate than the relatively simple operation for an ordinary esophageal hernia.

The esophageal hiatus hernia is repaired through an abdominal incision. The operation is a safe one and not particularly difficult. To show the conservatism with which the clinician has regarded this procedure in the past, our cases at the Cleveland Clinic may be briefly reviewed.

In a group of 51 cases—and I am limiting my discussion to esophageal hiatus hernia—only 2 were repaired surgically; 13 were considered inoperable because of age or presence of associated conditions (many of these cases occur in very old persons, who could not well tolerate surgery); in 18 cases the hernia was a small, symptomless, incidental finding requiring no treatment, and in the remaining 18 cases, though the patients were perhaps suitable for operation, for some reason or other none was prescribed in spite of the fact that in many instances symptoms were rather severe.

I became interested in trying to determine just why operation was not recommended in this last group. Some men said: "Well, it's such an extensive procedure," almost invariably assuming that it meant a transpleural approach. They failed to realize how simple it is to correct this condition.

That the symptoms of diaphragmatic hernia are very real and of great importance to the patient was indicated in a recent case which was repaired surgically. This patient had not only a large diaphragmatic hernia but also gallstones with biliary colic. The question having arisen whether we could operate for both conditions at the same time or whether we should do one or the other operation first, the patient was asked: "Which is bothering you most, the biliary colic or the hernia symptoms?" Her reply was: "The diaphragmatic hernia," and this she wanted corrected first since the symptoms were continuous and bothered her every day, whereas the attacks of biliary distress occurred only periodically and she knew pretty well how to cope with them. Fortunately we were able to do both operations simultaneously through a left rectus incision. The gallbladder was freely movable, and the operation was not technically difficult in any way.

The one important thing the surgeon has to determine before he operates for one of these hernias is the length of the esophagus. Is he dealing with a congenital thoracic stomach, with a short esophagus and the esophageal opening above the diaphragm? By injecting air into the stomach, he can usually outline the esophagus, determining that it is of normal length and that the hernia may be easily reduced.

Sidney A. Portis, M.D. (Chicago, Ill.): I have often said to my students that the diagnostic acumen of the gastro-enterologist is directly proportional to his experiences in the fluoroscopic room. Dr. Beilin has reported on a group of cases of diaphragmatic hernia, which, previous to the advent of the x-ray, were diagnosed only at necropsy or at operation. With roentgenological evidence, we can readily evaluate the clinical manifestations in these patients.

About three years ago, I reported to the Chicago Society of Internal Medicine three cases of so-called pseudo-angina pectoris, in which the symptoms so closely resembled the clinical syndrome of angina pectoris that clinical differentiation was impossible. In all of these cases the symptoms were due to a diaphragmatic hernia. The important clinical feature is the precordial distress which comes on immediately after eating and, as soon as the patient belches or vomits, is completely relieved. When one becomes conversant with this trend of symptoms, he may be justified in making a pre-roentgenological diagnosis of diaphragmatic hernia.

I have been impressed with the frequency of diaphragmatic hernia in patients over fifty years of age, found incidentally in the fluoroscopic room. Hiatal hernias are relatively common. It is only when they produce symptoms that they become of diagnostic importance. I have often felt that the frequency of hiatal hernias in elderly people may be associated with a relaxation of the diaphragm, the over-distention of the cardiac end of the stomach forcing it up through the diaphragm. This is especially apt to be found in patients with gallbladder disease.

Arthur R. Bloom, M.D. (Detroit, Michigan): I want to emphasize the point made by Dr. Portis, that hiatal hernias are encountered very, very frequently. Sometimes we see the same type of stomach without the hernia, that is, with the

greater curvature convex and the lesser curvature concave.

Among the cases of Dr. Beilin's series was one which especially interested me. That is the one with associated diverticula containing adenoma or papilloma. About two years ago I examined a woman who had been treated for secondary anemia. She had an extensive diaphragmatic hernia and large diverticula in the ileum and jejunum with negative shadows, presenting the appearance of polypoid growths. This case was almost an exact duplicate of Dr. Beilin's.

Irving I. Cowan, M.D. (Milwaukee, Wisconsin): I should like to emphasize a few points in regard to the method of roentgen study of diaphragmatic hernias.

In examination the use of a thick barium mixture or paste will enable one to determine more accurately the type of hernia existing. This finding is important in relation to the type of treatment that may be instituted. In addition, the use of the Trendelenburg position with rotation of the patient will in many instances facilitate the entrance of the hernia into the hernial sac. Also I believe the use of spot films during the procedure will aid in diagnosing a larger number of cases of diaphragmatic hernia.

Leo G. Rigler, M.D. (Minneapolis, Minnesota): All the discussants have mentioned the frequent occurrence of hiatus hernias. Five years ago Erickson and I reported, in a series of 5,000 patients who had gastro-intestinal symptoms, the presence of hernia in about 1 per cent. The incidence is about the same in our clinic at the present time. Most of the patients, however, had no symptoms referable to the hernia.

Dr. Eneboe and I also reported that in pregnant multiparae the incidence of hiatus hernia may be as high as 20 per cent during the period of pregnancy; in primiparae about 5 per cent. I am sure the factor of increasing abdominal pressure is a very important one.

D. S. Beilin, M.D. (*closing*): Spot roentgenograms are taken in all cases of diaphragmatic hernias that are of clinical significance. Small paraesophageal hernias producing no symptoms were not included in this series.

Roentgenographic Diagnosis of Vertebral Syphilis

PROFESSOR DR. MAX SGALITZER[1]
Istanbul, Turkey

THAT VERTEBRAL syphilis can be diagnosed roentgenographically and differentiated from other lesions involving the spinal column is demonstrated by three proved cases.

CASE REPORTS

CASE I: I. T., a 47-year-old man, gave a history of gradually increasing pain in the nape of the neck,

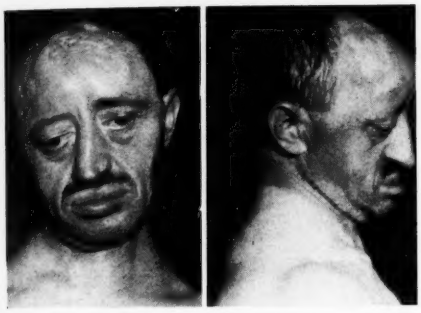

Fig. 1. Case I: Syphilis of the cervical spine.

dating back twenty-two years. The motility of the cervical column had decreased, and the pain, which radiated to both shoulders, had become so severe that the patient was on the verge of suicide. Drugs afforded little relief, and diathermy, mud compresses, sulphur baths, and treatment at various spas were without effect. Roentgen examinations had been made from time to time, showing a destructive process in the cervical column, suggesting to one examiner a possible osteomyelitis, to another a metastatic tumor.

For a year and a half the patient had been under the care of Dr. F. Redlich, by whom he was referred to the writer for roentgen examination. The internal organs had been found to be normal. The cervical column was fixed (Fig. 1) and extremely painful to the touch. The head could not be moved.

The roentgenogram (Fig. 2) revealed, in addition to severe osteosclerosis and hyperostosis of the entire cervical spine, extensive destruction of the bodies of the 3d, 4th, and 5th cervical vertebrae. The 4th cervical vertebra showed the greatest involvement, with large areas of destruction having irregular borders on its upper, lower, and ventral surfaces. In spite of the far-advanced destruction of the three cervical vertebrae—and especially of the 4th—there had been no collapse and no wedge formation of the vertebral bodies. The intervertebral disks were normal. The sclerotic transformation of the bone had extended in places to the arches of the affected vertebrae. This transformation was most marked in the arch of the atlas, which exhibited decalcified spots and areas of sclerosis, as well as a distinct change in shape. There was no

Fig. 2. Case I: Syphilis of the cervical spine; extensive destruction of the vertebral bodies accompanied by osteosclerosis and hyperostosis.

narrowing of the vertebral canal in the cervical section. The diagnosis was gummous vertebral syphilis. A Wassermann blood test was strongly positive. Antisyphilitic treatment was instituted (bismuth, arsenicals, iodides) and after more than twenty years of suffering the patient was relieved of his symptoms within a few weeks. There was no recurrence of pain after a year and a half. A roentgen examination after this period showed ankylosis of the cervical spine.

CASE II: J. J., a male of 36 years, had an ulcerated syphilis of the skull with widespread areas of bone destruction producing marked deformity.

[1]From the Roentgenological Clinic at the University of Istanbul. Director: Professor Dr. Max Sgalitzer. Accepted for publication in August 1940.

A striking restriction of facial expression was another consequence, due probably to injury to the nerves, especially the left facial. The head was held in extreme flexion. The neck was immovable, the nape strongly prominent. Besides the excessive syphilitic destruction of the cranium, syphilitic lesions were demonstrable in both humeri and the left ilium. The Wassermann test was strongly positive.

An x-ray examination of the cervical column, which was extremely sensitive and was held in fixed position, revealed even more profound changes (Fig. 3) than were observed in the preceding case.

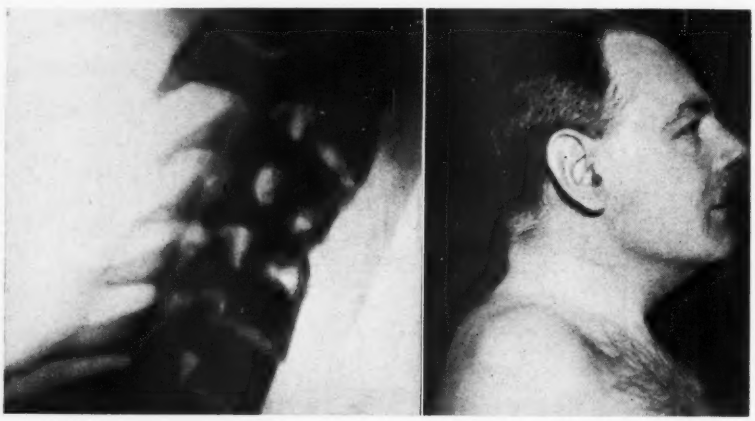

Fig. 3. Case II: Vertebral syphilis with extensive destruction of the upper and middle cervical spine. Simultaneous osteosclerosis prevents the affected vertebral bodies from breaking down.

The arch of the atlas was completely absent. Only a very narrow fragment of the body of the 2d cervical vertebra remained, corresponding to its dorsal portion. The ventral parts of the 3d, 4th, and 5th cervical vertebrae were absent and the remaining portions of their bodies had irregular borders on the ventral aspect. In spite of the extensive destruction of the vertebral bodies, however, and in spite of the fact that only a narrow fragment of the body of the 2d cervical vertebra remained, there had been no collapse and no wedge formation of the vertebral bodies, because all had undergone osteosclerotic transformation. The posterior wall of the pharynx was in immediate contact with the cervical column, so that the presence of an abscess could be excluded.

Energetic antisyphilitic treatment gave excellent results. The skull lesions healed completely, numerous sequestra being cast off. The excruciating pains in the cervical skeleton, of which the patient had complained, gradually disappeared. Eight years later roentgen examination of the cervical column showed complete healing of the destructive process, leaving an osseous ankylosis involving the upper and middle cervical vertebrae.

CASE III: M. K., male, thirty-seven years old, had malaria as a child but denied having any subsequent illnesses. Two other children of his parents died shortly after birth. He was himself married and had two children in good health. His wife's first pregnancy had terminated in abortion. For five months he had suffered violent pains in the right shoulder radiating to the right arm. A roentgen examination of the shoulder made elsewhere showed no abnormality, but certain changes were observed in the cervical column.

A roentgenogram of the cervical column (Fig. 4) now showed destructive changes of the body of the 6th vertebra. Sclerotic areas and a hyperostosis were observed and the upper and ventral surfaces were irregularly indented. The lower ventral portions of the body of the 5th cervical vertebra also exhibited a beginning sclerosis of the bony tissue, as did—to a slight degree—the upper ventral section of the 7th cervical vertebra, the margin of which projected like an exostosis. Both intervertebral disks of the 6th cervical vertebra were normal. The body of the 6th cervical vertebra had lost nothing or almost nothing in height. The axis of the cervical column showed a normal curvature. The arches of the affected vertebrae were unchanged.

On the basis of this picture a diagnosis of syphilis of the cervical spine was made. The patient denied a previous syphilitic infection, but the Wassermann test was strongly positive. A typical osteitis syphilitica was found in the diaphysis of the right humerus. Antisyphilitic therapy relieved the symptoms after three weeks.

DISCUSSION

The three cases of vertebral syphilis described here are alike in their localization in the cervical spine, the most profound changes being found in the upper and middle cervical vertebrae in Cases I and II. In all, *though one or more of the vertebral bodies had undergone extensive destruction, there was a complete absence of collapse, due to pronounced sclerosis and hyperostosis.* The axis of the cervical column remained almost normal and no wedging of the vertebral bodies occurred in spite of grave destructive changes. The areas of destruction, moreover, were mainly limited to the ventral portions of the vertebral bodies and showed irregularly indented borders. These changes were most striking in the second case, in which only a narrow fragment of the body of the 2d cervical vertebra remained and the ventral sections of the 3d, 4th, and 5th were completely destroyed; nevertheless, almost no diminution of the height of these four vertebrae was observed. In each case the intervertebral disks were preserved and in none was an abscess present, which would be recognized in the lateral view by displacement of the pharyngeal wall.

The bony change described here as characteristic of certain forms of vertebral syphilis is in accord with phenomena well known in pathological anatomy and frequently observed in the later stages of syphilis. The characteristic feature is the new bone formation accompanying destruction of the spongiosa and leading to hyperostosis and osteosclerosis. The syphilitic granulation tissue penetrates into the bone, forming rough cavities with indented borders and there spreads to form irregularly shaped channels; this accounts for the indented borders of the zones of destruction in the roentgen picture. With destructive spondylitis syphilitica, which is found chiefly in the cervical column, a gummous infiltration of the marrow takes place in one or several vertebrae. An abscess is almost never found.

Can vertebral syphilis be differentiated from other diseases? This question can be answered in the affirmative in the majority of the cases.

Differentiation from vertebral tuberculosis is not difficult. In contradistinction to vertebral syphilis, in which osteosclerosis is predominant, the tuberculous vertebra shows decalcified areas at an early stage. The body of the vertebra, if penetrated by tuberculous granulation tissue, will often collapse under the weight of the body early in the course of the disease and wedge formation will occur. The syphilitic vertebra, on the contrary, only exceptionally collapses, even in the presence of extensive destruction. Moreover, the favorite site of vertebral tuberculosis is close to the intervertebral disk, while vertebral syphilis is chiefly located in the ventral part of the vertebral body; the intervertebral disks are usually preserved in the presence of syphilis, while their destruction is a frequent symptom of tuberculosis. Cold abscesses are en-

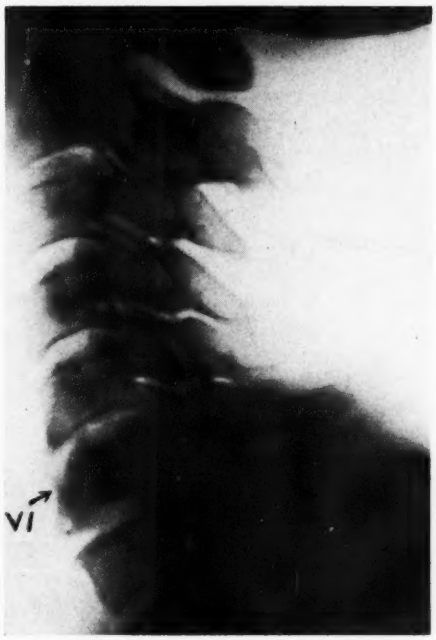

Fig. 4. Case III: Vertebral syphilis; destruction of the body of the 6th cervical vertebra. Simultaneous osteosclerosis prevents the affected vertebral bodies from breaking down.

countered in the majority of cases of vertebral tuberculosis, but are never found in vertebral syphilis. Vertebral syphilis usually extends over a greater part of the vertebrae than tuberculosis.

The differential diagnosis between vertebral syphilis and osteomyelitis does not offer any particular difficulty. If the osteomyelitic process has spread over the whole of the vertebral body, a high degree of decalcification will be observed; sclerotic condensation of the bone-substance, characteristic of syphilis, is found in osteomyelitis only in the stage of healing. Wedge formation and the presence of an abscess are both frequent in osteomyelitis.

Lymphogranulomatosis is also to be distinguished by the decalcification of the vertebral bodies, sclerotic transformation of the bone substance taking place only in the healing stage.

Osteoplastic tumor metastases usually spread over more extended sections of the vertebral column than syphilis or may involve the entire spine. A vertebral body that has been destroyed by tumor metastases often breaks down under the weight of the body, which—as has been pointed out—does not usually happen in syphilis.

Apart from the roentgen symptoms, the diagnosis of vertebral syphilis will be supported by the absence of a primary tumor in the breast, thyroid gland, prostate, ovaries, or bronchi. Obviously the diagnosis is strengthened by a positive serologic test, though it should not be forgotten that disease of spine due to other causes may be associated with syphilis. Rapid improvement following antisyphilitic therapy affords confirmation of the diagnosis.

The three cases of vertebral syphilis discussed here belong to the group *spondylitis syphilitica destructiva*, which chiefly involves the cervical spine. This disease is certainly not of frequent occurrence, but may undoubtedly be more common than is generally believed, often being undiagnosed because the symptoms are not sufficiently known. More frequent than the destructive form of syphilitic spondylitis is *spondylitis syphilitica simplex*, which is usually observed in the later stages of syphilis, only seldom in the secondary stage; it causes no changes in the vertebral skeleton and therefore offers no characteristic roentgen picture. It is usually characterized by more or less severe pain in the spinal column accompanied by swelling of some articulations which recede slowly during antisyphilitic treatment. In contradistinction to *spondylitis syphilitica simplex* the destructive form of vertebral syphilis offers, as has been shown, a quite characteristic roentgen picture, clearly distinguishable from other diseases of the vertebral column.

That knowledge of the roentgenological features of the disease is important is strikingly illustrated by Case I. This patient was sent from doctor to doctor, from hospital to hospital, from health-resort to health-resort for twenty years, without obtaining relief. After the correct diagnosis had been made on the basis of the roentgen picture, causative therapy was initiated and complete recovery promptly ensued.

SUMMARY

Three cases of syphilis of the spinal column are reported. The lesion is characterized radiologically by definite symptoms, so that it can be differentiated from other diseases of the vertebral column.

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New X-Ray Laboratory of the National Bureau of Standards[1]

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DURING THE past ten years, excitation voltages used in x-ray therapy have been gradually raised from the 200-kv. level generally established in the '20's to about 1,000 kv. at the present time. Today there are, in this country, over 100 plants operating at 400 kv. and some dozen or more at potentials between 400 and 1,000 kv. With this increase in excitation voltages there has been a corresponding demand for extension of x-ray dosage standards as well as new studies in the clinical application of the radiation administered. The problem of the establishment of suitable standards in this voltage range has been delegated by the various societies and committees to the National Bureau of Standards, as a logical extension of its facilities and experience in the lower voltage range.

The Bureau, however, lacked both space and equipment for this work. Accordingly, about two years ago, Congress appropriated the sum of $500,000 to provide these facilities along with related facilities for high-voltage investigations and testing by the Electrical Division. Of this sum roughly half was put into the building and the remainder into building equipment, and high-voltage and x-ray equipment. This paper will present a brief description of the x-ray equipment and that part of the building relating particularly to the x-ray section. The Electrical Division's part of the building and equipment has already been described by Silsbee.

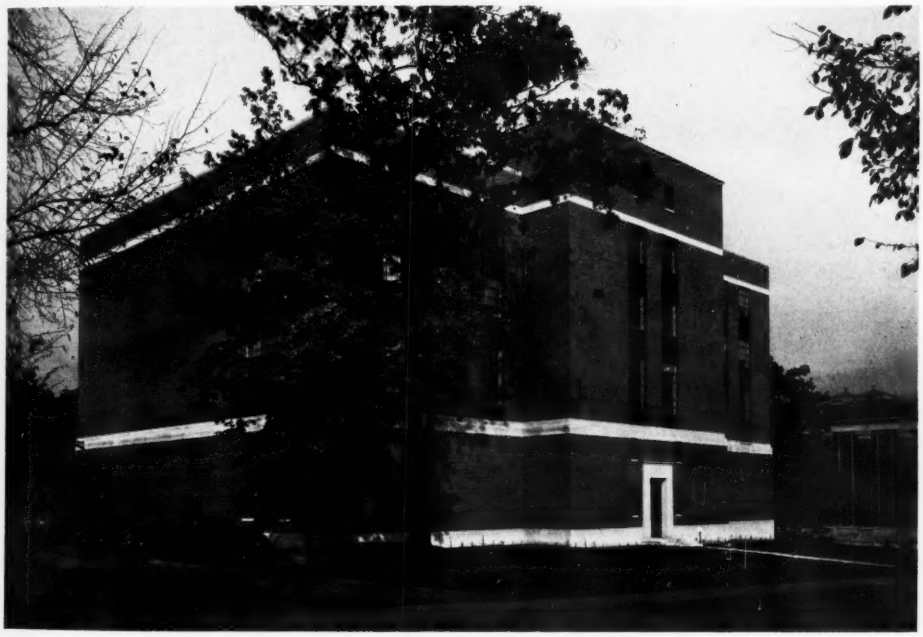
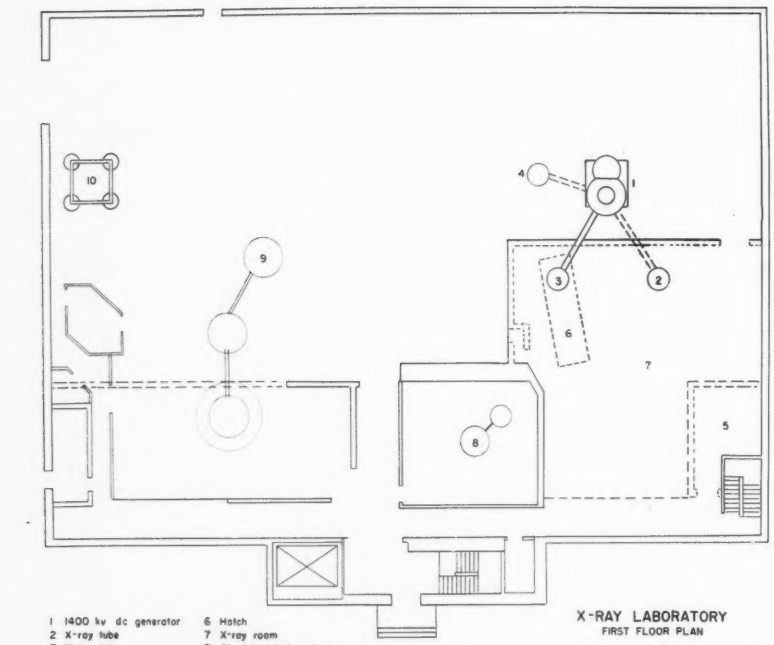
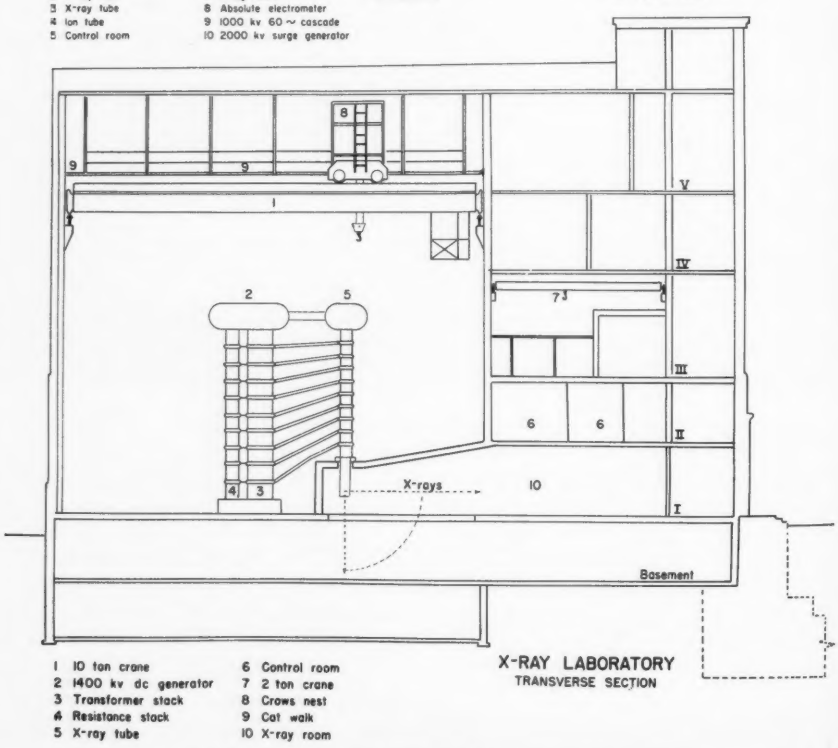


Fig. 1. Outside view of high-voltage and x-ray laboratory, National Bureau of Standards.

The building (Fig. 1) is about 135 feet

[1] Presented before The Radiological Society of North America at the Twenty-sixth Annual Meeting, at Cleveland, Ohio, Dec. 2–6, 1941.





Figs. 2 and 3. First floor plan and laboratory cross-section.

long by 96 feet wide and 80 feet high. Across the front are 5 floors 30 feet wide, for laboratories above ground level, while the remaining two-thirds of the volume, in the back, comprises a transformer bay having a clear space 130 × 65 × 60 feet high, as shown in Figure 2. In addition to the floors above ground is a basement laboratory beneath part of the x-ray end of the building and a sub-basement for air-conditioning equipment. The entire building space is allocated, according to function, about equally between the x-ray section and high-voltage measurements section.

To the rear of the building is a one-story annex, 33 × 76 feet, which contains all of the motor-generator equipment, storage batteries, primary switchgear, and other equipment, such as compressors, pumps, etc., which produce noise or vibration. This is to maintain a very low background noise level to facilitate location of corona on equipment in the main building.

The building is entirely air-conditioned for the primary purpose of disposing of toxic gases produced by corona, and maintaining a low relative humidity in order to permit operation during summer months. The air-conditioning equipment is so designed as to permit either recirculation or complete exchange of air. Complete electrical distribution services are provided throughout the building. The primary distribution point for power is in the annex. Trunks from this are led from a main switchboard on the first floor, from which distribution lines are led to subpanels, two on each floor throughout the building. In addition, all of the boards have direct interconnections. Leading out from the plug panels on each floor is a series of outlet boards by means of which ready distribution of power may be made through the plug board. Through this distribution system it is possible to obtain power directly from the A.C. or D.C. mains from the main bureau power plant, from storage batteries, or from any one of a half dozen special motor-generator sets located in the annex.

Figure 2 shows the general arrangement of equipment on the first floor. At (*1*) is the main high-voltage transformer-rectifier for exciting any one of three tubes (*2*, *3*, and *4*). Projecting into the main bay is a low ceiling concrete cubicle atop which rest the two 1,400-kv. x-ray tubes (*2* and *3*), the targets of which are beneath the cubicle, thereby permitting safe occupation of the remainder of the main bay while x-rays are being used. The third tube, not yet built, will rest on the main floor at *4* and extend into the basement laboratory. This will be a positive ion tube. At the other end of the building is indicated the location of some of the Electrical Division's equipment.

A cross-section of the laboratory is shown in Figure 3. Here are shown the transformers (*3*) and tubes (*5*). Beneath one of the tubes (*3* in Fig. 2) is an opening of 6 × 20 feet into the basement, to permit unobstructed measurements of x-rays over an angle of 0–90° with respect to the cathode beam. This is covered with a metal floor when not in use. X-rays are normally directed as indicated and thus completely confined to the space under the cubicle.

The transformer bay has a metal roof, copper-covered walls and metal-grilled concrete floor all thoroughly bonded together. While designed primarily to assure of fixed potentials throughout the space, it also serves to cut out radio interference from spark-overs. Extending across the bay is a 10-ton electric crane (*1*), 45 feet above the floor. Around the entire room, 10 feet below the ceiling, is a cat-walk (*9*) to facilitate rigging operations. In addition there is a crow's-nest (*8*) atop the crane carriage by means of which any portion of the ceiling may be reached with ease. The room is without windows and hence total darkness can be obtained.

The transformers (Fig. 4) are designed to give a constant potential of 1,400 kv. and a maximum current of 15 ma. with a ripplage of 0.08 per cent per ma. (Charlton and Hubbard). The transformer unit is

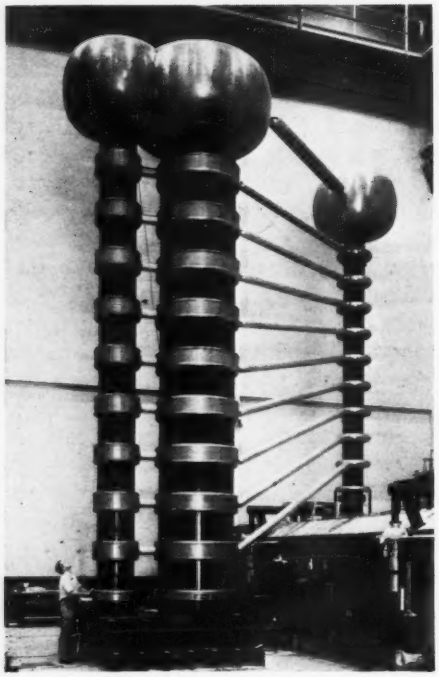


Fig. 4. The 1400 kv. transformer rectifier.

composed of 10 identical units of 140 kv. each, stacked one atop another and rigidly bolted together. The bolting sections are covered with adequately rounded corona bands. The whole stack is topped with an aluminum corona cap about 11 feet across and 4 feet high. Each individual unit contains a 140-kv. transformer, two rectifiers and filament transformers, two capacitors, and a polarity reversing switch operable from without the tank. The complete stack may thus be operated with the top cap at either a positive or negative potential. Each unit has a Herkolite bottom and walls, and a steel top through which insulated leads run to the adjoining unit.

Adjoining the main transformer stack is a Herkolite tube, 22 inches inside diameter, and the full height of the stack, which contains a 1,400-megohm wire resistor which serves as a voltmeter multiplier for accurate voltage measurement. In addition there is mounted on the ceiling, directly above the transformer, a Behr generating voltmeter for voltage control. The x-ray tubes seen at the right are of the multisection type with 12-inch diameter glass sections about 22 inches long. Surrounding this is a Herkolite supporting tube atop of which rests a corona cap 6 feet in diameter and 4 feet high. Connection between tubes and transformer is made with 10 telescoping aluminum tubes spaced at 140-kv. intervals, thus assuring uniform potential distribution along the tube. These connecting tubes are swung from one x-ray tube to the other by means of an operator in a boatswain's chair suspended from the crane.

The entire equipment is operated from within a control room at the second floor level and located behind the "cubicle" space (*6* in Fig. 3). The main control station is designed along "unit panel" lines to permit future changes if necessary. The equipment is completely electrically interlocked, to prevent the possible starting of any component until all preceding operations are satisfactorily completed. All interlock operations are indicated with lamps and annunciator drops. In the center panel are 4 potentiometer indicators for generating voltmeter, resistance voltmeter, target current, and x-ray emission, respectively. Complete control is achieved by a field rheostat for voltages and a key for tube current, both conveniently located for use by a single operator.

For protection against x-rays, concrete is used exclusively except for the doors, which are lead. In addition, advantage has been taken of the softening of x-rays by scattering, and the control rooms have accordingly been located in such positions as to necessitate at least one 90° scattering of the radiation from the target. The attendant softening of the radiation through the scattering has been sufficient to permit major economies in the protective installations. Electrical safety has been provided by having all doors operate safety switches tied into the interlock circuit so that the equipment cannot be turned on while a door is open. In order to prevent someone from opening the

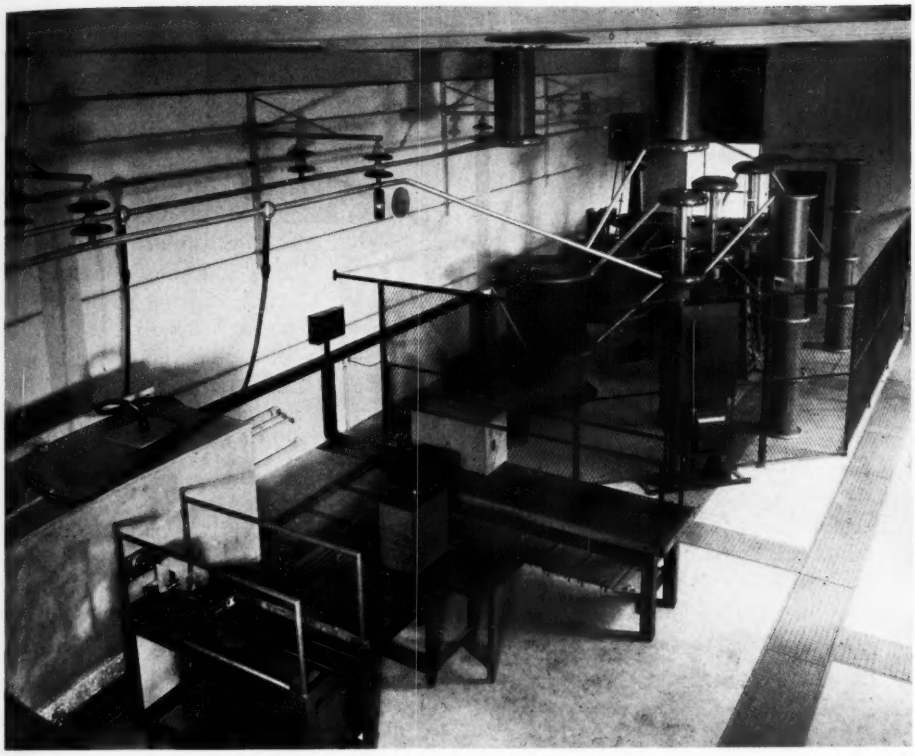


Fig. 5. View of 400 kv. x-ray room.

doors, thereby turning off the equipment, they are held closed magnetically while the equipment is in operation. Across the main hall and surrounding any high-voltage equipment on the other floors are grounded metal fences, the gates of which are tied into the electrical interlock system. The cubicle walls and roof and the walls separating the main bay from the laboratory floors are 12 inches thick, and the walls of the control room 16 inches thick. Measurements have shown that at no point of normal occupation by personnel does the stray radiation level exceed one-tenth the generally accepted tolerance dose level.

The other laboratory floors contain lower voltage equipment and auxiliary services. The second floor level in addition to the control room has a balcony designed for experimental vacuum systems, a high-frequency induction heater, and a hydrogen firing furnace for preparation of x-ray tube parts.

In a room 70 × 30 feet on the third floor is the low-voltage standardization equipment (Fig. 5). This room is also equipped with a one-ton traveling crane to facilitate the handling of heavy equipment. Voltage is supplied by a very flexible transformer-rectifier operable up to 400 kv. This employs the continuously evacuated rectifier as used in our older equipment (Taylor, Singer, and Charlton). Available here are three x-ray tubes, a 400-kv. sealed-off tube (not yet installed), a 220-kv., 20-ma. oil-cooled tube, and a 180-kv., 4-ma. thin glass air-cooled tube, the last two being mounted on a track to permit easy movement between different sets of standards.

The control equipment on the third floor is patterned exactly after that em-

ployed for the 1.4 million-volt equipment, and is located in a concrete cubicle having walls 10 inches thick to provide the necessary protection from stray radiation.

On the fourth floor are the offices in the front and laboratory space in the back, in which is contained a low-voltage set (150 kv., D.C.) and a high-power water-cooled tube operated from a halfwave generator at 220 kv. and 30 ma. This latter is to be used in connection with liquid ionization studies. The fifth floor has a large open shop space about 30 × 50 feet, containing light machine tool equipment and facilities for welding, soldering, etc. Also located on this floor is a small chemistry and glass-blowing room equipped with exhaust blowers apart from the air-conditioning system. A large dark room contains three sets of developing spaces, of which one is to be used primarily by the x-ray section, one by the high-voltage section, and the third jointly for such work as special developing and enlarging work. The remainder of the floor is taken up by the Atomic Physics Laboratory.

At the time of writing, the installation of the million-volt equipment has been completed and the equipment is now in operation. The 400-kv. equipment on the second floor has just been completed and standardization work is about to be undertaken once more. The equipment on the fourth floor has largely been moved aside in order to make space for some urgent war preparation work.

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National Bureau of Standards, Washington, D. C.

The Effect of High and Low Body Temperatures upon the Growth of Irradiated Mouse Sarcoma 180[1]

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A RECENT STUDY (1) showed that tumor regression following irradiation was markedly increased by daily injections of distilled water or hypotonic Locke-Ringer solution into the tumor. Swelling of the cells generally followed. It is our belief that the increase in radiosensitivity of a tissue may be due to increase of circulation or a decrease of the osmotic pressure of the surrounding extracellular fluids. It is extremely important to learn the rôle of fluids in the reactions of irradiated living organisms.

This work was planned to determine the significance of water balance in relation to irradiated tumors. The problem was approached by studying the combined effects of roentgen radiation and fever, and roentgen radiation and cold upon transplantable tumors.

EFFECT OF HIGH BODY TEMPERATURE UPON THE GROWTH OF IRRADIATED TUMORS

It is believed that in fever there is a considerable disturbance in the water balance of the body. Thirst exhibited by patients is interpreted as evidence of a demand for water by the tissues.

Fever may be induced in animals by various methods: administration of drugs (2, 3), vaccine (4), toxins (5), or glucose (6), and exposure to short and ultra-short radio waves (7–30). In order not to complicate our task, we chose the latter method.

Studies on the action of ultra-short radio waves upon cancer date back only to 1928, when Schereschewsky succeeded in producing therapeutic effects upon mouse, rat, and chicken tumors. A number of papers have appeared since. The literature cited (7–30), however, does not offer consistent conclusions. Some writers believe that the degree of effectiveness varies with different tumors and different wavelengths. Others interpret the healing influence as a heat effect, while still others found that wavelengths varying between 2.8 to 29.0 meters had no inhibiting effect upon animal tumors.

As a preliminary study, we have determined the effect of ultra-short radio waves on mouse sarcoma 180 *in vivo*. The source of the ultra-short waves was a Siemens Ultratherm apparatus, having a wavelength of 6 meters.

Special attention was given to the amount of food ingested, water intake, and urine excretion of the irradiated mice. A careful record was also kept of the physical condition, body weight, body temperature and duration of life.

A basal diet, consisting of cracker meal (National Biscuit Co.) 80 per cent, whole milk powder (Klim) 19 per cent, and irradiated yeast (Fleischmann) 1 per cent, was selected for the present study. A dough was prepared with 50 parts of the basal diet and an equal amount of water, and a weighed amount of the food was given daily. All animals were confined in metal cages with screen bottoms, and distilled water was given in unlimited amounts.

Pairs of animals (Rockland albino mice) bearing tumors from one to seven days old were placed in glass crystallizing dishes, 97 mm. in diameter, 50 mm. in height, 1.5 mm. thick, each having a wooden partition in the center to prevent contact of the two animals. A catalin cover, having holes for ventilation, was placed over each dish. The dish containing the animals was placed on a fabric table between two parallel electrodes (Schliephake glass electrode 4 inches in diameter), 7 cm. from the top electrode and 5 cm. from the bottom

[1] From the Chemical Laboratory, Memorial Hospital, New York City. Accepted for publication in July 1940.

electrode, as shown in Figure 1. At first, the filament voltage was brought up to 22 volts and quickly tuned to the resonance point. The voltage was then rapidly decreased to the desired value. Generally, the machine was operated at 18 and 17 volts across the electrodes. Under these conditions severe symptoms appeared after five minutes' exposure at 18 volts or twenty minutes' exposure at 17 volts. The animals all perspired profusely and were unable to walk upright. Death followed if the exposure was prolonged. Mice in this condition felt warm to the touch, and rectal temperatures immediately after death ranged from 42.5 to 43.0° C., occasionally reaching 44° C. Many animals, however, survived an increase of body temperature to 43° C. The elevated temperature dropped to normal in a few minutes when the animals were removed from the apparatus. The treatment was generally repeated daily for two to six days.

In the winter of 1937 and summer of 1938, 18 sets of experiments were carried out involving a total of 96 tumor-bearing animals. About an equal number of non-irradiated tumor-bearing mice were used as controls.

Single or repeated exposures of the entire body of the tumor-bearing animals to waves of 6 meters in length gave 10 per cent cures. The remaining 86 treated tumors continued to grow normally and killed the hosts, or ulcerated and disappeared. On the other hand, non-irradiated controls showed 6 per cent spontaneous regression. The difference is thus too small to be significant.

Rectal temperatures of the treated animals reached from 40 to 43° C., while the same animals immediately before treatment and non-treated controls showed rectal temperatures of 36 to 40° C., generally 37.2° C.

The thermal death times for mouse sarcoma 180 were determined by immersion of tumor fragments in Locke-Ringer's solution at *p*H 7.4 (31). The bottles containing tumor fragments were occasionally shaken. The growth capacity of this tumor was completely destroyed by six hours' immersion at 37° C. and three hours' immersion at 42° C. After immersion in various blood sera for twenty-four hours at 37° C., the following results were obtained. Seventy per cent inhibition was found with normal guinea-pig serum, 75 per cent inhibition with normal rat serum, 80 per cent inhibition with normal mouse serum, 90 per cent inhibition with normal human serum, and 100 per cent inhibition with normal rabbit serum.

Rohdenburg and Prime (32) found that the thermal death times *in vitro* for mouse sarcoma 180 were three hours at 42° C. and one-half hour at 46° C., while the thermal death times determined by Jares and Warren (33) for this same tumor were much longer: fifteen hours at 41.5° C. and eight hours at 42° C. This discrepancy in results may be due to the unphysiological environment of the tumor tissues. In the former case the fragments of tumor tissue were suspended in Ringer's solution in test tubes, without *p*H control, and the tubes were not shaken during the experiments. In the latter case the tumor tissues were suspended in Locke's solution at a *p*H 7.4 and shaken during the experiments.

Feeding experiments with young adult mice, irradiated and non-irradiated, showed that individual animals consumed about 4 gm. of basal diet daily. Each irradiated mouse consumed 8.0 c.c. of distilled water daily compared with 5.5 c.c. for non-irradiated controls. This figure represents the total calculated water consumption including that from the food ingested, which amounts to 50 per cent of the weight of the food.

Investigations upon the action of ultra-short radio waves on the growth of x-ray-irradiated tumors were next made.

As before, tumors (about seven days old and 5 to 8 mm. in diameter) of mouse sarcoma 180 were irradiated *in vivo* with a dose of 500, 750, or 1,000 r in the manner previously described (34). Within one to two hours after irradiation, the tumor-bearing animals were exposed to the ultra-

short radio waves, having a filament potential of 18 or 17 volts, for five to thirty minutes. In general, the ultra-short radio wave treatment was repeated on five or six successive days. These animals exhibited a marked reaction to the ultra-short waves. All perspired profusely throughout the period of treatment. The rectal temperatures were from 40 to 43.3° C. The results are presented in Table I. In this table are also included results obtained in a previous x-ray study (x-rays alone) with a much larger number of experiments. The present study included 10 groups of experiments, involving a total of 60 tumor-bearing animals; 20 tumors were irradiated with 500 r, 20 with 750 r, and 20 with 1000 r.

Table I shows clearly that the number of regressions was definitely increased when tumors were irradiated with x-rays *in vivo* followed by repeated exposure to ultra-short radio waves.

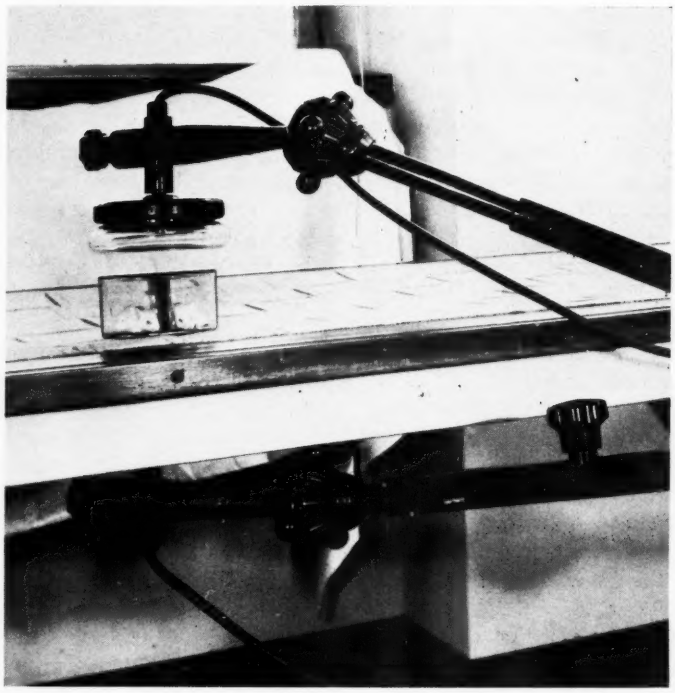

Fig. 1. Position of glass dish containing animals for exposure to ultra-short radio waves.

TABLE I.—EFFECT OF ROENTGEN RAYS AND ULTRA-SHORT RADIO WAVES UPON THE GROWTH OF MOUSE SARCOMA 180

Dose in Roentgens	Tumor Regression X-Rays Alone	X-Rays + Heat
500	3%	20%
750	15%	45%
1,000	50%	75%

EFFECT OF LOW BODY TEMPERATURE UPON THE GROWTH OF IRRADIATED TUMORS

It is well known that decrease in temperature retards chemical as well as physiological reactions. Consequently we were interested in trying the effects of low temperature on the viability of irradiated tumors.

In the course of preliminary observations, it soon became evident that when the environmental temperature of mice is reduced to −10° C., all the animals die

within four hours. When, however, the temperature of the environment is increased to 3 to 5° C. the mice survive the experimental period of four weeks.

Accordingly, a number of healthy young adult male albino mice, weighing from 22 to 25 gm., were divided into two groups, one to be kept at a very low temperature and the other at room temperature. Metal metabolism cages containing from 5 to 10 animals were placed in an electric refrigerator, the cooling chamber of which measured 32 × 72 × 38 inches. It was provided with heavy glass windows and had an outlet for adequate ventilation and humidity (Fig. 2). The refrigerator was kept at 3–5° C. The temperature of the animal room during July and August of 1939 was from 25 to 32° C., averaging 29° C.

As before, the animals were given a basal diet consisting of cracker meal 80 per cent, whole milk powder 19 per cent, and irradiated yeast 1 per cent, with as much distilled water as they would take.

The results obtained from this study may be briefly summarized as follows. Animals confined at low temperatures became inactive and tended to huddle together most of the time in order to keep warm. Some gained weight slightly during the first two weeks, but maintained their weight thereafter; others showed no gain. Each mouse consumed about 3.9 gm. of food and about 2.5 c.c. of water daily. This represents the total calculated water consumption including that from the food ingested which, as stated above, amounts to 50 per cent of the weight of the food. The daily urine excretion was about 1.7 c.c. Each control mouse kept at room temperature consumed about an equal amount of food daily (4.2 gm.), but the daily water intake was much greater, 5.5 c.c. Daily urine excretion per mouse in the control group was about 0.30 c.c. This figure is low because of rapid evaporation in the metal metabolism cage due to high room temperature. Furthermore, the daily urine excretion was not constant. In order to obtain more uniform results, a similar experiment was conducted in a constant refrigerated room, 9 × 10 × 13 feet, at 21° C. It was found that the average daily urine excretion per mouse was 0.41 c.c.

The rectal temperature of mice kept in the refrigerator was from 34.4 to 38° C., generally 36° C.,[2] while the rectal temperature of the same animals before placing them in the refrigerator was from 37 to 39.3° C., generally 37.9° C. The experimental animals regained normal health and vigor quickly when returned to room temperature after a long confinement in the refrigerator and gained body weight normally. None of the animals developed pneumonia. Two groups of experiments were conducted, involving a total of 40 mice.

As part of the present study an attempt was made to ascertain whether a low environmental temperature is capable of exercising a destructive action upon transplanted tumors.

The young adult mice were kept in a refrigerator at 3–5° C. for one to three days prior to tumor implantation. Fragments of mouse sarcoma 180 were inoculated subcutaneously into these animals and they were left in the refrigerator for about four weeks. Growth of the sarcoma in animals kept at the temperature of 3–5° C. was retarded during the first week, but the transplants grew normally thereafter (Fig. 3). The low temperature influenced neither the tumor takes (100 per cent) nor regressions. There was, however, a slight increase in the life of these tumor-bearing animals. At room temperature, mouse sarcoma 180 kills animals in two to three weeks from the time of transplantation. When the tumor-bearing animals are kept at 3–5° C., they generally live four weeks. This study included 4 groups of experiments, involving a total of 60 tumor-bearing animals.

[2] During the experimental period of four weeks the temperature of the refrigerator rose several times to about 9° C. As soon as this was noticed, the temperature control was adjusted in order to bring the temperature down to 3–5° C.

Three tumors from the preceding study were removed from the animals at the end of one, two, and three weeks, respectively, and non-necrotic fragments were inoculated into normal animals kept at room temperature. The number of tumor takes and the rate of growth of the tumor grafts removed from the cooled animals were practically the same as for those taken from controls. This indicates that the malignant character of tumor cells of sarcoma 180 cannot be altered by cold treatment. This study included three groups of experiments, involving a total of 60 mice.

In order to secure further information concerning the activity of cooled tumors *in vivo*, we made the following experiments.

Animals bearing seven-day-old mouse sarcoma 180 were placed in a refrigerator at about −10° C. After the death of these animals they were kept continuously from forty-eight to seventy-two hours at about 3° C. At the end of these periods the tumors were removed from the animals and tumor fragments were inoculated into normal animals in the usual way. The results showed that the normal transmissibility of mouse sarcoma 180 was not changed by keeping the tumor in the host for two to three days at 3° C. The growth of tumors was delayed, however, in the first week, while thereafter growth was generally normal. There was no evidence

Fig. 2. Metal cages containing animals in a large refrigerator in the course of prolonged cold treatment.

of stimulation of tumor growth. This indicates that cold probably does not act as a selective agent, killing susceptible cells and leaving vigorous tumor cells unharmed.

It may be of interest to mention that the growth capacity of mouse sarcoma 180 was completely destroyed when the tumor remained in the host for eighteen hours at 22° C. after the host had been killed by a blow or by ether anesthesia. The autolysis of tumor tissue is responsible for the inactivation.

We next determined the effect of low temperature upon the growth of x-ray-irradiated mouse sarcoma 180.

As before, when tumor grafts had grown for about seven days in animals and had attained diameters of 5 to 8 mm., they were irradiated with a dose of 500, 750, or 1,000 r in the manner previously described. Within three hours after irradiation, the tumor-bearing animals were placed in a refrigerator kept at 3–5° C. and allowed to remain continuously for about four weeks. The animals were given as much food and water as they would take. The results are presented in Table II. This study included 5 groups of experiments, involving a total of 42 tumor-bearing animals whose tumors had been irradiated; 11 tumors were irradiated with 500 r, 16 with 750 r, and 15 with 1000 r. In this table are also included results obtained in a previous x-ray study (x-rays alone) comprising a much larger number of experiments.

TABLE II: EFFECT OF LOW BODY TEMPERATURE UPON THE GROWTH OF IRRADIATED MOUSE SARCOMA 180

Dose in Roentgens	Tumor Regression	
	X-Rays Alone	X-Rays + Cold
500	3%	0
750	15%	12%
1,000	50%	27%

As may be seen from Table II, the number of tumor regressions decreased when tumors were irradiated with x-rays *in vivo* followed by continuous confinement of the hosts in a refrigerator at 3–5° C. In other words, the radiosensitivity of the tumor is decreased by reducing the body temperature of the hosts. The rectal temperature of these mice was one to four degrees below the normal temperature of 37.9° C.

DISCUSSION

In comparing the results obtained between the irradiated tumors followed by repeated exposure to ultra-short radio waves (Table I) and those kept in continuous confinement in a refrigerator (Table II), it will be noted that the number of tumor regressions is much greater in the warmed animals than in the cooled animals. In the former case, both the body of the animal and its tumor are in a physiologically active state, while in the latter case they are more or less in a dormant state. It is interesting to note also that the water intake and amount of urine excretion differ greatly between the two groups, although the food consumption is almost the same (Table III).

TABLE III: WATER BALANCE IN X-RAYED MICE SUBJECTED TO CHANGES IN TEMPERATURE

Animal Subjected to	Body Temperature (degrees C.)	Food Intake (gm.)	Water Intake (c.c.)	Urine Excretion (c.c.)
Ultra-short radio waves	43	4.0	8.0	0.25
Refrigeration, 3–5° C.	36	3.9	2.5	1.7
Room temperature, 21° C.	38	4.2	5.5	0.41

Table III shows clearly that the body temperature of animals treated by ultra-short radio waves was greatly elevated above normal. The animals all perspired profusely throughout the period of ultra-short radio wave treatment, tending thereby to lose water and salt. On being returned to their cages they evidenced thirst, drinking considerable amounts of distilled water. A group of animals kept at the low temperature of 3–5° C. consumed very little water but excreted a much larger amount of urine than those subjected to ultra-short radio wave treatment. The difference between the average water intake of the warmed animal dur-

ing twenty-four hours, 8.0 c.c., and the cooled animal, 2.5 c.c., is significant. The difference in urine excretion, 0.25 c.c. for warmed animals and 1.7 c.c. for cooled animals, is also significant.

It is interesting to note that the body temperature of animals kept continuously in a refrigerator at 3–5° C. for four weeks was relatively high, 34.4 to 38° C. When the temperature of the environment was reduced to about −10° C., however, all the animals died in about four hours. The body temperatures of these animals did not vary greatly from the normal temperature until shortly before death.

In the course of the investigation a study was made on the resistance of mouse sarcoma 180 to extreme cold *in vitro*.

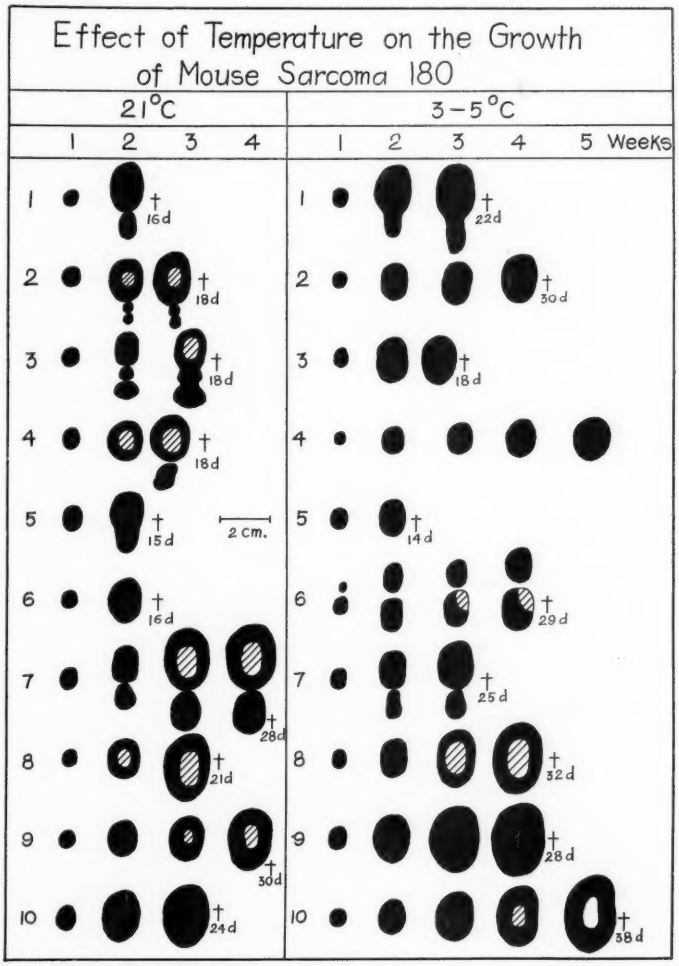

Fig. 3. The growth of sarcomas in mice kept at a temperature of 3–5° C. was slightly retarded during the first week after transplantation, but was normal thereafter. The low environmental temperature alone influenced neither tumor takes nor tumor regression.

Ten or more small pieces of tumor tissue, each weighing about 6 mg., were placed on a piece of filter paper slightly moistened with a Locke-Ringer solution at *p*H 7.4 in a celluloid pill box having a diameter of 2.4 cm., height 1.1 cm., and thickness

0.4 mm. Parafilm was wrapped around the pill boxes to prevent entrance of CO_2. The boxes were placed in a thermos bottle containing pieces of solid CO_2. At the end of definite intervals of time, they were removed from the solid CO_2; the tissues were thawed rapidly at room temperature and inoculated into normal mice. The results are presented in Table IV.

TABLE IV: RESULTS OF TRANSPLANTING MOUSE SARCOMA 180 KEPT AT −70° C.

Number of Tumor Fragments	Duration of Frozen State (hours)	Growth of Transplants (per cent)
10	1	80
20	2	90
10	4	90
20	24	85
10	48	90
10	72	80
10	120	80
10	168	80

As may be seen from Table IV, the growth capacity of mouse sarcoma 180 was only slightly inhibited when tumor fragments were kept at −70° C. Under normal conditions, the percentage of tumor takes is 100. The growth of tumors was markedly delayed in the first week, but thereafter growth was normal in all cases. The deterioration of tumor cells was the same whether they were kept one hour or seven days in the frozen state. This indicates that the injurious action of an extremely low temperature on tumor cells is due to the initial rapid freezing, which causes formation of small ice crystals, and not to keeping them in the frozen state for long periods (35, 36, 37).

SUMMARY

1. The combined effects of 200 kv. roentgen rays and fever, and 200 kv. roentgen rays and cold, on mouse sarcoma 180 *in vivo* have been studied.

2. The number of tumor regressions was definitely increased when tumors were irradiated with x-rays *in vivo* followed by repeated exposure to ultra-short radio waves.

3. The number of tumor regressions was decreased when tumors were irradiated with x-rays *in vivo* followed by continuous confinement of the hosts in a refrigerator at 3–5° C.

4. The growth of mouse sarcoma 180 in animals kept at 3–5° C. was retarded. The low environmental temperature alone had no influence upon tumor takes or upon tumor regression.

5. The growth capacity of mouse sarcoma 180 was almost entirely unaffected when tumor fragments were kept in the frozen state at −70° C. for seven days prior to transplantation.

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Memorial Hospital, New York.

The Effect of Shockproof Cables and Condensers on Two-Valve Half-Wave Rectification[1]

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VARIOUS CHANGES in roentgen therapy equipment during the past few years have afforded an opportunity for comparing the quantity and quality of radiation produced by several types of rectifying circuits and particularly for observing the effect of the capacity of shockproof tubes and cables and of additional capacity, in the form of condensers, on two-valve half-wave rectification. Since all the machines were in regular use for treatment, it has not been possible to make all the changes in the circuits for which data might have been desired, and since the oscillographic equipment was not available at the time some of the circuits were in use, their voltage wave forms could not be obtained.

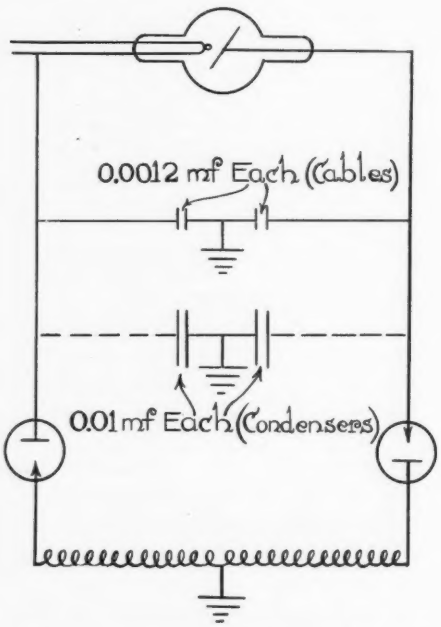

Fig. 1. Diagram of the two-valve half-wave rectifying circuit. The two 0.01 microfarad condensers could be connected to the circuit or disconnected from it.

The combinations of rectifying circuits, types of tubes, and types of connections between the rectifier and tubes which have been available are listed in Table I. Since the x-ray tube current was not always the same, the total intensity in roentgens per minute would not be expected to be the same; therefore, comparisons must be made on the basis of roentgens per minute per milliampere. All measurements were made in free air at 16 inches (41 cm.) target-ionization chamber distance. Peak kilovolts were determined with both a vacuum tube peak voltmeter and an oscillograph (9) for the combinations of valve rectification and shockproof tubes, and with a sphere gap for the other combinations; sphere-gap values have been in general a few per cent lower than the values obtained by the other two methods. The filtration used with the various combinations, although listed as 6 mm. aluminum, has not been exactly the same, since with the air-cooled tube it was all provided by added filters, while with the shockproof tubes the 6 mm. includes the assumed value of 0.5 mm. aluminum inherent in the tube and tube housing. The tube currents are given in average milliamperes since they were measured by a direct-current milliammeter in the high-tension circuit.

The intensity in roentgens per minute per milliampere is seen from Table I to vary more than 100 per cent, and it is seen that a variation is produced by a change in the type of rectification used, in the type of tube, or in the type of connection between the high tension and the tube. Although, as stated previously, peak volts and filtration may not have been exactly the same in all instances, and, as shown by Taylor and his associates (7), different circuits show

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TABLE I: QUANTITY OF RADIATION PRODUCED BY VARIOUS COMBINATIONS OF RECTIFYING CIRCUITS, X-RAY TUBES AND TYPES OF CONNECTIONS
(130 kv.p., 6 mm. aluminum filter, 16 inches distance)

Rectification	Tube	Connections	Ma.	r/min.	r/min./ma.
Mechanical	Air-cooled	Reels	5	20.2	4.04
	Shockproof A_1	8 ft. cables, 1.125 in. diameter	8	18.5	2.31
Two-valve half-wave	Air-cooled	Reels	5	21.3	4.26
	Shockproof B	20 ft. cables, 1.375 in. diameter	6	15.2	2.53
	Shockproof A_2	20 ft. cables, 1.125 in. diameter	6	13.7	2.28
Two-valve half-wave with condensers	Shockproof A_2	20 ft. cables, 1.125 in. diameter	6	27.6	4.60

less variation in output per effective milliampere than per average milliampere, it is believed that nearly all this variation is due to differences in the shape of the voltage wave (effective voltage) applied to the tube. Some variation in output due to tubes can be expected because of different target angles and different relationships of tube current and voltage for constant filament current, and slight variations may be attributable to the size and shape of the focal spot and differences in the thickness of the tube wall, thickness of oil, and exit window of the tube housing. Shockproof tubes A_1 and A_2 were from a different manufacturer than shockproof tube B, but the difference in output from tubes A_2 and B is believed to be due primarily to the difference in the size of the cables. The form of the voltage wave applied to the tubes from the same rectifier differed; that from tube B indicated less capacity in the rectified circuit than that from tube A, a difference which would be expected since the capacity per foot of cable would decrease as the cable diameter increased.

All the data given in Table II were obtained with the same two-valve half-wave rectifier and x-ray tube. The high-tension circuit is shown diagrammatically in Figure 1. The amount of capacity in the rectified circuit was varied by connecting two condensers of 0.01 microfarad each to the circuit or disconnecting them from it, and the voltage applied to the primary of the transformer was adjusted so as to obtain the desired peak voltage across the x-ray tube for each value of the tube current. Voltage wave forms, as traced from the oscillograph, for each peak voltage and current combination listed in Table II are shown in Figure 2 (without the two 0.01 microfarad condensers) and in Figure 3 (with the two 0.01 microfarad condensers).

In Figures 2 and 3 the solid lines show how the voltage across the x-ray tube varied with time, and the dotted lines show the variation of the voltage across the terminals of the transformer. Since the machine gives half-wave rectification, the voltage across the x-ray tube should coincide continuously with the transformer voltage during the useful half cycle (represented by transformer voltage above the zero kilovolt line) and remain at zero during the blocked half cycle (represented by the transformer voltage below the zero kilovolt line) if there were no capacity in the circuit (assuming that the voltage drop across the rectifying valves is negligible).

When there is capacity (condensers) in the circuit between the valves and the x-ray tube, the condensers are charged to the peak potential of the transformer during the useful half cycle. From that instant the potential of the condensers, hence the potential across the x-ray tube, decreases at a rate dependent upon the size of the condensers (their capacity) and the rate at which the charge on the condensers is removed, until the instant the transformer and condenser potentials are equal; the condensers are then charged by the transformer until the peak voltage of the transformer is reached and the cycle is completed.

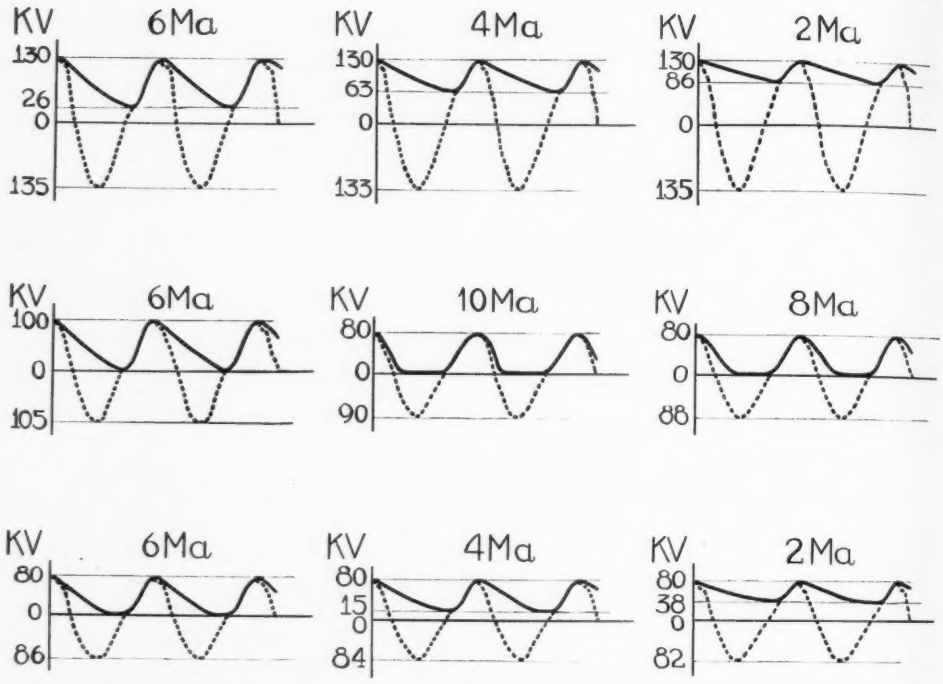

Fig. 2. Oscillograph tracings of the x-ray tube voltage (solid lines) and the transformer terminal voltage (dotted lines) for different milliamperes at three peak voltages from a two-valve half-wave rectifier with 20-foot (6.1 meter) shockproof cables.

Since in Figure 2 the tube potential never exactly follows the transformer potential, it must be assumed that there is some capacity in the circuit. Calculations made from data taken from the curves show that the value of the capacity in the circuit is almost exactly equal to the manufacturer's value for the capacity of the two cables (60 micromicrofarads per foot, or two condensers in series of 0.0012 microfarads each).

If the capacity of the condensers is small and their rate of discharge great (x-ray tube current large), the potential across the condensers may decrease almost as rapidly as the potential of the transformer, and the wave form of the potential across the tube approaches that of the useful half wave of the transformer as shown in Figure 2 for 80 kilovolts and 10 milliamperes. As the rate of discharge of the condenser is decreased the wave form of the potential across the tube departs more and more from that of the transformer and eventually approaches constant potential; true constant potential would be obtained if there were no current flowing through the tube, hence no discharging of the condensers. If the tube current remains constant and the amount of capacity in the circuit is increased, the difference between the peak and minimal voltages of the condensers is decreased, as is shown by comparing corresponding curves of Figures 2 and 3.

If the capacity of the circuit and the tube current remain constant, the kilovolt difference between the peak and minimal voltages remains nearly constant regardless of the value of the peak potential; hence the percentage variation in voltage is greater for low peak potential than for high peak potential. It is seen, for example, from Figure 3 that for a tube current of 2 milliamperes the difference for 130 peak kilovolts is 8 kilovolts or 6.1 per cent, and the difference for 80 peak kilovolts is 7 kilovolts or 8.7 per cent. This might not

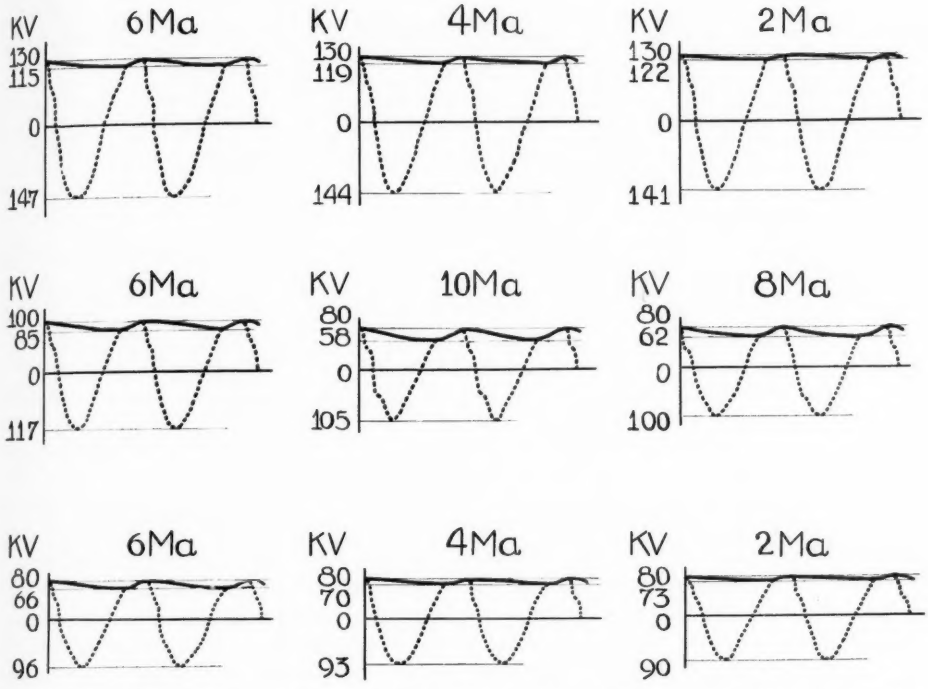

Fig. 3. Same as Figure 2 except that two 0.01 microfarad condensers have been connected to the rectified circuit.

be true for all x-ray tubes and depends on how the tube current varies with tube potential. If the current decreases as the potential decreases, the rate of discharge of the condensers would not be constant, and in some instances the difference between peak and minimal potentials might be a constant percentage of the peak potential instead of a constant kilovolt difference, or it might be neither. That the current was practically constant over a wide range of voltages for the tube used in these experiments is shown by the fact that the decreasing voltage is represented by practically a straight line in Figures 2 and 3, indicating a constant rate of discharge of the condensers. This could be shown, also, by the fact that with a constant setting of the x-ray tube filament current the tube current remained practically constant regardless of the voltage applied to the tube when the circuit included the condensers.

The potential generated in the secondary of the transformer bears a constant relationship to the potential applied to the primary of the transformer. If current is flowing through the secondary, part of the potential is used to drive the current through the coils of the secondary and is not available for use in driving a current through the x-ray tube or to charge the condensers. For this reason the potential at the terminals of a transformer in a two-valve half-wave rectifying circuit is greater during the unrectified half cycle than during the rectified half cycle. This difference will not be the same for all transformers. In general it increases as the magnitude of the current increases. This is shown clearly in Figures 2 and 3. That there may be exceptions to this rule due to the characteristics of a transformer is shown in Figure 2, in which the potential generated by the transformer was the same for both 6 and 2 milliamperes when the tube was operated at 130 peak kilovolts.

Since the intensity of radiation generated by an x-ray tube at any instant is propor-

TABLE II: QUALITY AND QUANTITY OF RADIATION PRODUCED BY A TWO-VALVE HALF-WAVE RECTIFIER WITH 20-FOOT SHOCKPROOF CABLES WITH AND WITHOUT TWO 0.01 MICROFARAD CONDENSERS CONNECTED TO THE RECTIFIED CIRCUIT

(All measurements at 16 inches distance in free air)

Kv.p.	Filter, mm.	Ma.	No condensers			With condensers		
			HVL mm.	r/min.	r/min./ma.	HVL mm.	r/min.	r/min./ma.
130	6 Aluminum	6	0.255 Cu	13.7	2.28	0.320* Cu	27.6	4.60
		4	0.250 Cu	10.8	2.70	0.325 Cu	18.8	4.70
		2	0.275† Cu	7.12	3.56	0.330 Cu	9.64	4.82
100	2 Aluminum	6	2.74 Al	17.0	2.83	3.18 Al	34.2	5.70
80	0.5 Aluminum	10	1.06 Al	53.5	5.35	1.13 Al	83.3	8.33
		8	1.05 Al	42.1	5.26	1.15 Al	68.4	8.55
		6	1.03 Al	30.3	5.05	1.17‡ Al	53.2	8.87
		4	0.97§ Al	20.0	5.00	1.18 Al	36.2	9.05
		2	1.01 Al	13.8	6.90	1.19 Al	18.4	9.20

* Equal to 130 kv.c. with 0.15 mm. Cu filter.
† Equal to 110 kv.c. with 0.15 mm. Cu filter.
‡ Equal to 80 kv.c. with 0.45 mm. Al filter.
§ Equal to 70 kv.c. with 0.45 mm. Al filter.

tional to the square of the voltage at that instant, the quantity generated in a longer period, or roentgens per minute, will be proportional to the mean of the squares of the instantaneous values of the voltage during that period, or proportional to the square of the effective voltage. But since for different wave forms there is no constant relation between peak and effective voltages, there is no constant relation between peak voltage and quantity of radiation. Hence, as shown in Table II, there may be very great differences in roentgens per minute for the same peak voltage if there are such changes in the wave form of the voltage as shown in corresponding curves of Figures 2 and 3, even if the milliamperes, filtration, and distance are kept constant. The ratio of maximal and minimal instantaneous intensities during one cycle of the voltage wave can be determined from the ratio of the squares of the maximal and minimal voltages. Such a calculation shows that at 130 peak kilovolts and 6 milliamperes the minimal intensity is only 4 per cent of the maximum when the condensers are not in the circuit, but is 78 per cent of the maximum when the condensers are in the circuit. The actual variation in intensity may be more than that calculated if the voltage varies over a wide range, since the filters will absorb a greater proportion of the lower voltage radiation than of the higher voltage radiation. On the other hand, if the tube current varies with the voltage, the variation of the intensity may be less than that calculated because of the lower current at the lower voltages.

It is often assumed, at least for any one x-ray machine, that the intensity of the radiation will be directly proportional to the tube current (constant roentgens per minute per milliampere) if the peak voltage is kept constant. The data in Table II show that this is not necessarily true. If the wave form of the voltage is altered by a change in tube current there will be a change in roentgens per minute per milliampere due to the change in effective voltage. Since the curves in Figures 2 and 3 show that changing the milliamperes produces a change in the voltage waves, a variation in intensity per milliampere such as is shown in Table II should be expected. Without the condensers in the circuit there is a marked change in voltage wave form (Fig. 2) with a change in current and a marked change in the intensity per milliampere, but with the condensers in the circuit there is only a slight change in voltage wave form (Fig. 3) with a change in current and a small change in intensity per milliampere.

The quality of a beam of x-rays is determined primarily by the voltage across the tube and the filtration in the path of the beam. Again it is the shape of the voltage wave form, and not the peak voltage,

which is of importance. This is shown in Table II. Changing only the tube current produces some change in quality, shown by the half-value layers, but a greater change in quality is produced by changing the capacity (connecting or disconnecting the condensers) in the circuit. The effect of the condensers on the quality is shown also by comparing complete absorption curves with standard absorption curves from a constant potential generator (5). Thus the hardest radiation produced with 130 peak kilovolts without the condensers (with 2 milliamperes tube current) corresponds to that produced by 110 kilovolts constant potential, whereas that from 130 peak kilovolts with the condensers (with 6 milliamperes tube current) corresponds to 130 kilovolts constant potential, a difference of 20 kilovolts. For 80 peak kilovolts the quality with and without condensers in the circuit corresponds to that produced by 80 kilovolts constant potential and 70 kilovolts constant potential, or a difference of 10 kilovolts. In general, a change in voltage wave form that produces an increase in the intensity per milliampere increases the half-value layer, although this is not always true (Table II). These differences in quality may not be of much clinical significance, but it is entirely possible that greater differences might be found on other machines and at other peak voltages. The variations in quality in some cases might be sufficient to produce considerable differences in the amount of backscatter and depth doses if these were calculated from data on the basis of peak voltage and filtration rather than on measured quality values such as half-value layers.

Although the voltage wave forms presented here are all from the two-valve half-wave rectifier, it is logical to assume that the variations in roentgens per minute per milliampere for the other conditions listed in Table I, as well as similar data in the literature, are primarily attributable to differences in voltage wave form. These differences in wave form are due to both the rectifier itself and the amount of capacity in the high-tension system between the rectifier and the tube. The great effect on both quantity and quality of radiation that may be produced by the small capacity of shockproof cables has been a surprise to some radiologists when they have first installed shockproof equipment. Rogers (4) investigated the effect on intensity of cables of various lengths, using several types of rectifiers, and reported that with the Villard, self-rectified and one-valve circuits there was very little effect; that with a four-valve full-wave circuit there was a small increase in the intensity as the length of the cable was increased; and that on a two-valve half-wave circuit there might be either an increase or a decrease in the intensity, depending on the length of cable and the tube current. He suggested that the effect is due to a change in the voltage wave form produced by the capacity of the cables. Nurnberger (3) has reported that both the intensity and the half-value layer were increased when a shockproof tube and cables were used in place of a Universal air-cooled tube on a mechanical rectifier.

It has been known for many years that the voltage wave form did have a marked effect on the quantity and quality of radiation, but satisfactory equipment for recording voltage wave forms has not been readily obtainable until recent years. Chamberlain and Newell (1), in 1924, devised a method for obtaining some idea of the voltage wave form from ionization measurements made over different portions of the voltage cycle, which showed that there was a difference in the voltage wave form for two machines having radiation intensities varying more than 100 per cent. More recently Newell (2) has described a spectrographic apparatus with which data can be obtained for determining part of the voltage wave form supplied to an x-ray tube.

Taylor and his associates (5, 6, 7, 8) have shown that, although the output of radiation might vary more than 100 per cent and the quality be different from different types of rectifiers operating at the same peak voltage and tube current, there was

very little variation if the effective voltages were kept constant; the variations were reduced still further if the tube currents were measured in effective instead of average milliamperes. They found that the effective voltage, and therefore the radiation, were both affected by changing the amount of capacity in the high-voltage system. They also pointed out that some of the variation is due to the characteristics of the tube itself, particularly the relationship between tube current and voltage.

The data presented here emphasize again the well known fact that neither the quantity nor the quality of the radiation from a tube can be calculated from the peak voltage, current, and filtration, and that practically any change in the equipment may produce unexpectedly large changes in the radiation output. If any change is made in equipment or technic involving the x-ray machine, a complete recalibration should be made unless previous tests have shown that such changes have no effect on the radiation output. Major changes, such as type of rectification or the addition of condensers to the circuit, are usually expected to have considerable effect on the output; but even minor alterations, such as changing tubes, cables, or tube current, may produce unexpected effects. A two-valve half-wave rectifying circuit is probably affected more by such changes than most of the circuits in common use.

CONCLUSIONS

The effect of the capacity of shockproof cables and condensers on the voltage wave form from a two-valve half-wave rectifier is shown by oscillograph tracings for different milliamperes at several peak voltages. These different voltage wave forms produce changes in the intensity and the quality of the radiation. Shockproof cables, the presence or absence of other condensers, and different tube currents may produce changes of more than 100 per cent in the number of roentgens per minute per milliampere, as well as changes in the quality of the radiation.

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DISCUSSION

Lauriston S. Taylor, Ph.D. (Washington, D. C.): It is particularly fitting that we should learn something about the question of the effect of cable capacity on tube output. There is a great deal of old equipment in the hands of radiologists which is good for something, and which they are tending to remodel. They have the transformers; they will add shockproof cables and tube heads and will get rid of mechanical rectifiers, open tube holders, etc. I suspect that most people have regarded this problem in the same casual way that I have, assuming that adding cable capacity to a half-wave rectifier will probably increase the tube output.

I was surprised to find that that is not necessarily the case at all. The fact that a small capacitance can influence wave form to such a point as actually to decrease the intensity of the output is an exceedingly interesting fact and it only goes to show that if a radiologist is going to have some of his old equipment remodeled by the use of cable equipment, it will probably pay him to have either the service man or a consultant make a fairly careful analysis of the system to see just what the result will be. It may prove better to start anew with something that is designed properly in the first place.

That is the important feature, I believe, of Dr. Williams' contribution. I hope that he will be able to continue this work and carry it out with other forms of equipment, so that it will be a little easier to generalize; and perhaps put in printed form information which will better prepare the physicist and radiologist to undertake this remodeling of x-ray equipment.

Robert R. Newell, M.D. (San Francisco, Calif.): I am much interested in what Dr. Williams has said, and a little chagrined because it is something that anyone should have been able to prophesy if he had looked at the matter with a clear eye and unprejudiced mind.

We had a half-wave outfit, recently replaced, which we ran for more than fifteen years, which had the reputation of being the slowest x-ray machine in the world. We made two sets of investigations to find out why, and thought we had an explanation. I now think that the effect reported by Dr. Williams was really responsible.

We investigated the wave form of the machine by an analysis of the x-ray output and our first analysis, to which Dr. Williams has referred, was done by observing the hardness of the x-ray during various parts of the cycle. Later we made a strobospectrograph which consisted of a spectrograph, that is a rock salt crystal, which threw the spectrum through a slit onto a film, which was kept rotating with a synchronous motor. Thus we got spots of x-ray on the film during the part of the cycle when the x-ray was coming from the tube and the short wave limit indicated the maximum voltage on the tube at each portion of the cycle. The trouble is, of course, that during those parts of the cycle when the voltage is so low as to give a negligible quantity of x-ray through the wall of the tube no record is obtained on the strobospectrogram. There might, however, still be several milliamperes running through the tube which show on the milliampere meter but do not produce any x-ray. That is the portion of the cycle that is destroying the efficiency of the x-ray outfit.

When a capacity of 0.005 microfarad is introduced, the machine is converted into a constant potential machine. We have presumably already ruled out that solution on account of expense.

Another possible solution would be to shunt the valves with a resistance so that during the inverse cycle the current could leak back around the condensers and reduce the voltage on the condensers very rapidly instead of very slowly, and one might adjust the value of these high resistances so that the inverse voltage might pile up to, say, half-voltage or something of that sort, and still destroy most of this inefficiency due to x-ray current running out of the cables at voltage insufficient to produce x-rays.

John Russell Carty, M.D. (New York City): A consideration of cable failure is another aspect of this interesting problem. Cable failure is generally attributed to excessive wear, faulty manufacture, or overload, particularly along the lines of excessively high voltage. I wonder if some of these failures, cannot be attributed to a high-frequency resonance phenomenon.

As I recall, in Dr. Williams' circuit diagrams there was inductance as well as capacity. These two elements are present in every high-tension circuit where shockproof cables are used, the inductance, of course, being the transformer, while the capacity is represented by the condenser effect of the cables themselves. Under these conditions where the factors are favorable, it seems to me that high frequency might well be produced.

Marvin M. D. Williams, Ph.D. (*closing*): The presence of capacity in the high-tension circuit, whether it is in the form of shockproof cables or condensers, greatly increases the possibility of having high voltages produced by high-frequency oscillations, particularly if there is a slight amount of gas in either the x-ray or a valve tube. We saw indications of such high-frequency oscillations on several occasions before we inserted a resistance of 60,000 ohms between each condenser and the rest of the circuit. It is probable that high-frequency oscillations are occasionally the cause of cable failure.

Two other methods of increasing the output from these machines were considered. Shorter cables could have been used, but the gain in output would have been small. Two more valves could have been added, giving full-wave rectification, but this would have been more expensive than adding the condensers and would not have given as great an increase in output. Since the machines operate at only 130 kvp., the condensers are not very expensive.

Addendum. The possibility of shunting the valves with a resistance to allow the cable capacity to discharge through the transformer instead of through the x-ray tube, as suggested by Dr. Newell, has been investigated on the model x-ray machine[1] as well as from theoretical considerations. Both methods show that the value of the resistances could be chosen so that the voltage wave form applied to the x-ray tube during the useful half cycle would be very much improved, but if the value of the resistances is low enough to give a good useful voltage wave, the peak inverse voltage applied to the tube would be about 80 per cent of the useful peak voltage. The machine then would have many of the disadvantages of the so-called self-rectified machines. Another disadvantage of shunting the valves with a resistance might be the difficulty in determining the actual x-ray tube current, because milliammeters placed in either the high-tension system or the grounded lead from the transformer would be affected by the charging and discharging condenser currents on both half cycles. This difficulty of accurately measuring the current would be present also with self-rectified equipment using shockproof cables unless one end of the x-ray tube were grounded. The difference between the meter reading and the actual tube current probably would be small in most instances, since short cables are generally used, and the percentage difference would decrease as the tube current was increased.

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[1] Electric model of an x-ray machine, shown by Marvin M. D. Williams in the Scientific Exhibits at the Twenty-sixth Annual Meeting of the Radiological Society of North America, Cleveland, Ohio, December 2 to 6, 1940.

CASE REPORTS

Os Intermetatarseum: Unusual Variant[1]

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Os intermetatarseum is described as an accessory bone of rather rare occurrence (1, 2, 5). Almost all of the cases thus far recorded have been rather diminutive osseous bodies lying usually between the bases of the first and second metatarsals. Sometimes the bone appears as spindle-shaped, projecting a half or a third of the distance to the heads of the other metatarsals.

Fig. 1. Os intermetatarseum: antero-posterior view of foot. The articulation of the accessory fourth metatarsal with the proximal phalanx is here distinctly shown. Note that this toe has only two phalanges. There is evident, also, a Freiberg's disease of the second metatarsal head, with broadening of the base of the articulating phalanx.

One well known authority writes in a recent text (5): "The os intermetatarseum is a small accessory bone lying between the bases of the first and second metatarsal bones; it is sometimes attached to the second metatarsal bone and projects in such a way as to suggest the appearance of a rudimentary metatarsal bone. It is seen only rarely and does not give rise to symptoms."

A search of the literature tends to confirm this observation as to symptoms, though in one case occurring at the New York Orthopaedic Dispensary and Hospital, such a bone occurring between the first and second metatarsals was considered to play an etiologic rôle in the production of a hallux valgus (6).

The case here presented is of interest because of the unusual situation of an os intermetatarseum; it appears in the space between the fourth and fifth metatarsals, articulates distally with the phalanx, the "true" fourth metatarsal head appearing to be distinctly extra-articular, and tapers to a small proximal end, which appears free. The "true" fourth metatarsal articulates with the tarsus at its base. It would appear that the "true" fourth metatarsal lies below, the accessory metatarsal above. A further anomaly is present in that the fourth toe appears to have only two phalanges.

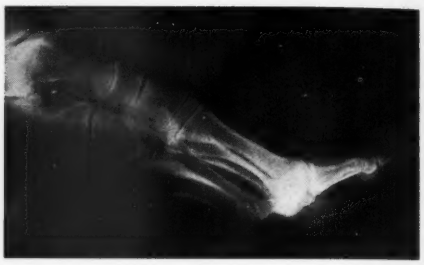

Fig. 2. Os intermetatarseum: lateral view. The articulation of the accessory fourth metatarsal with the proximal phalanx is again seen. The "true" fourth metatarsal appears to lie below, the accessory above.

On physical examination the foot appeared to show some increase in thickness in the region of the fourth metatarsal. The fourth toe was shorter than the corresponding toe on the opposite foot. A callus was present over the fourth metatarsal head region on the plantar surface of the foot.

The patient, who was a woman fifty years of age and who had been sent for examination to rule out fracture in a case of sprained ankle, gave only an ambiguous history of any previous symptoms referable to the foot. She was questioned further because of an incidental finding of Freiberg's disease (deforming metatarsophalangeal osteochondritis) in the second metatarsal head. If there had ever been an acute phase in the latter condition, the history gave little clue to it.

Relative to Freiberg's disease, Hauser (5) makes the comment: "The condition is believed to be the result of mechanical strain and abnormal repeated

[1] From the Department of Radiology, West Suburban Hospital. Accepted for publication in September 1940.

traumas that occur at the heads of the second and third metatarsal bones. It is usually seen in cases of pes valgoplanus associated with metatarsus latus.

"Since the second metatarsal bone is generally the longest, it is exposed to increased weight bearing and trauma."

In the case here described, one cannot help regarding as somewhat logical the conclusion that the distortion of the metatarsal arch which seems to have been produced by this os intermetatarseum may very likely have been etiologic in the production of a Freiberg's disease of the second metatarsal head.

SUMMARY

A case is here illustrated in which an os intermetatarseum occurs between the fourth and fifth metatarsals; its head appears to articulate with the proximal phalanx of the fourth toe, while the base of the "true" metatarsal articulates with the tarsus. The fourth toe is further anomalous in that it has only two phalanges.

The inference presents itself that the distortion of the metatarsal arch produced by the os intermetatarseum may have been etiologic in the Freiberg's disease, which was present in the same foot.

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Hypodermoliths: Report of a Localized Case[1]

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A startling and unusual condition which receives little notice in textbooks or medical literature is that form of calcinosis which Piersall (1) has called hypodermoliths. In 1933 he reviewed the literature, discussed the chemistry and pathology of the disease, and presented two cases. I cannot amplify his comprehensive presentation of the disease but add another rather spectacular case to the limited number already reported.

The patient, R. L. A., was a C.C.C. enrollee, white, age 21. His family and previous personal history were negative. On May 30, 1939, a beam fell on his left thumb. The injury was not serious enough to cause him to stop working, but the thumb remained sore and about two weeks later began slowly to swell. It became more painful and nodules appeared in the swollen area. The condition progressed up to the time of admission to the hospital, Aug. 31, 1939.

A lesion was also present on the inner side of the right thigh. This the patient believed followed a bruise but could not say when it began. The appearance of the knee on admission was not noted, but on Sept. 9, under 1 per cent novocaine, a calcareous deposit was removed from the subcutaneous tissue on the inner side of the thigh just above the knee, for biopsy. It consisted of small rounded deposits of a putty-like substance which became hard on exposure and drying. The specimen was sent to The Army Medical Museum for examination and diagnosis.

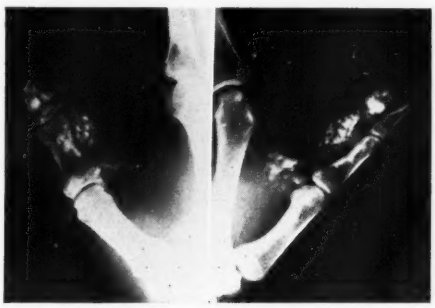

Fig. 1. Postero-anterior and lateral views of left thumb, showing hypodermoliths in the soft tissue. U. S. Army Medical Museum negatives Nos. 69,958 and 69,957.

The pathological report (Accession No. 65182) was as follows: "Specimen consists of a piece of tissue irregular in shape, 1 cm. in diameter, in the center of which is a white material, putty-like in consistency. Section consists chiefly of a mass of débris staining darkly with hematoxylin and generally globular in form. As the tissue has been through xylol, these masses cannot be pure lipoid but may be a phospho-lipoid with calcium added. The mass has a fibrous capsule with no epithelial lining.

"Diagnosis: Calcium or other mineral deposit of undetermined nature, in fibrous capsule. Etiology obscure."

The local laboratory findings were essentially negative.

On Oct. 15, four small incisions were made in the left thumb and several of the calcareous deposits were removed from the subcutaneous and deeper tissues. Many of the masses were inaccessible and were left *in situ*. The synovial sheaths were not incised and did not appear to be involved.

No other areas of involvement were found by either physical or roentgen examination. In January 1940, the right knee was red and enlarged but not hot or tense. Multiple nodules were felt on

[1] Accepted for publication in June 1940.

Fig. 2. Calcium deposits in soft tissues of right knee and thigh. U. S. Army Medical Museum negative No. 69,959.

both sides of the patella, extending upward in the subcutaneous tissues. There was a draining sinus at the site of operation which had broken down after remaining closed for about two months. The sinus constantly exuded a whitish discharge, which in appearance and consistency suggested evaporated milk. It contained some cellular detritus and multiple fine amorphous granules and tended to solidify on exposure. The sinus eventually closed.

On April 3, 1940, the condition was still progressing slowly, with calcareous nodules breaking through the skin of the thumb from time to time. Another operation was attempted under local anesthesia and as many of the deposits were removed as possible without undue mutilation of the thumb. These incisions healed in about two weeks and the patient left the hospital.

Roentgen Findings, Sept. 1, 1939: Films of the left thumb show multiple opaque, discrete, closely packed, rounded shadows in the flexor soft parts extending from the web nearly to the tip of the thumb. In the proximal phalangeal segment the shadows resemble the acini of a mulberry. The density of the shadows is that of calcium. Bones of the thumb appear normal except for slight osteoporosis. The tumors are not connected with the bones but are subcutaneous and in the soft tissues (Fig. 1).

Dec. 16, 1939: Films of the right knee region show numerous rounded structureless deposits in the subcutaneous tissues on both sides of and anterior to the patella, extending upward in the fascia on the inner side of the thigh for 15 cm. The bones are not involved and there are no calcified bodies in the joint. Later films show slight extension of the involved area, with the calcium becoming more granular in appearance around the knee (Fig. 2).

Comment: The diagnosis was elusive in this case. Only after reading Piersall's comprehensive article did we finally ascertain the true nature of these peculiar calcium tumors. Hypodermoliths appear to be a localized form of calcinosis and have been called epidermal stones, gout stones, calcareous gout, and petrifaction of the skin. Scleroderma is a related disease.

The etiology is obscure and the hypotheses put forward are as diverse as those offered for other tumor formations. There is some disturbance of the calcium metabolism without involvement of the parathyroids or abnormality of the serum calcium and phosphorus. Köhler (2) states that in certain cases the subcutaneous calcium deposits are a sign of tuberculosis of the skin.

Treatment is unsatisfactory. Surgical removal of calcareous deposits is occasionally indicated but since the concretions sometimes develop in traumatized tissue, further trauma does not appear to be the logical treatment. Recurrence was prompt in the case recorded here. Kennedy (3) reported from the Mayo Clinic the case of a child with calcinosis and scleroderma treated by a ketogenic diet, with almost complete disappearance of the subcutaneous calcifications over a period of four years. Kennedy also discusses the pathology and theories of the disease.

As to prognosis, the localized type of calcinosis is much less serious than the generalized. The latter type may cause very severe disability. The localized calcifications may be annoying but may remain stationary for many years without impairing the general health unless sinuses with chronic infection develop following the extrusion of hypodermoliths. The use of a ketogenic diet in this disease complex is encouraging; and a few patients are reported to have recovered without treatment.

Note: Acknowledgement is made to the Medical and Surgical Services of the Station Hospital, Fort McPherson, for assistance in working up this case.

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EDITORIAL

Howard P. Doub, M.D., Editor

John D. Camp, M.D., Associate Editor

Gastritis and Antral Narrowing

The scientific study of gastritis was begun by Beaumont, whose epoch-making work in observing the open stomach of Alexis St. Martin laid the foundation for subsequent studies on gastric physiology and pathology. Robertson[1] quotes Beaumont to the effect that after excessive liquor or food consumption, and in states of undue excitement, fear, and anger, the villous coat became red and dry. Sometimes eruptions with red pimples were seen on the gastric lining. Beaumont noted with interest how much abnormal change could be present inside the stomach without any external change or sign of functional aberration.

The gradual development of the technic of roentgen examination, together with the use of the Wolf-Schindler flexible gastroscope, has given us improved methods for studying the physiology of the living stomach and the pathologic changes occurring in it. Similarly the safety of present-day gastric surgery has made available to us abundant freshly resected material for histologic study. These advances have given us a new interest in inflammatory conditions of the stomach.

Gastritis originates in the mucosa and may be limited to that portion of the stomach or may involve any of the coats. In general two types are recognized—the hypertrophic and the atrophic. Schindler and Ortmayer[2] recognize a third type—the superficial—although they admit that the latter may be an early phase of atrophic gastritis, as the two frequently exist together.

Superficial gastritis, as its name implies, tends to involve only the superficial layers of the mucosa. It may be an acute condition secondary to dietary indiscretion and is usually self-limited. Gastroscopically red, apparently hyperemic spots are seen, which are occasionally associated with small mucosal erosions. These show little evidence of edema or elevated margins. A thick gray mucus is observed clinging to the walls.

In atrophic gastritis there are atrophy and thinning of the mucosa so that the blood vessels of the submucosa may be seen through it.

Hypertrophic gastritis is characterized by infiltration of the gastric mucosa. The mucosal folds are thickened and swollen and because of their induration are difficult to obliterate by moderate pressure. Mucosal and submucosal ulceration is common. While this ulceration has given rise to conjectures as to a possible etiologic relationship to peptic ulcer, most authors believe that the ulceration of gastritis usually tends to disappear without the development of peptic ulcer. In cases of resected gastric ulcer, however, an accompanying gastritis is often described by the pathologist.

In the general roentgenologic study of gastritis it has been assumed that the size and appearance of gastric folds are of great diagnostic importance. These findings, however, may be misleading, for it has been demonstrated that the submucosa has greater influence on the width of these folds than has the mucosa. Thus in the presence of extensive mucosal changes overlying an intact submucosa, the gastric folds may still appear normal. On the other hand, under certain conditions enlarged broadened folds may be of considerable importance. Large folds which are not obliterated by pressure, and which have a stiffened, immobile appearance, are of significance. Variation of the normal mucosal fold pattern may also be important.

A more significant but rather rare roentgenologic sign is the demonstration of hyperplastic granular changes which vary from small rounded defects in the mucosal relief to larger defects which may simulate gastric polyposis. The demonstration of erosions on the mucosal folds is a diagnostic sign of decisive character

[1] Robertson, H. E.: Ulcerative Gastritis and Residual Lesions. J.A.M.A. **112:** 22–27, Jan. 7, 1939.

[2] Schindler, R., and Ortmayer, M.: Classification of Chronic Gastritis with Special Reference to Gastroscopic Method. Arch. Int. Med. **57:** 959–978, May, 1936.

but unfortunately is rarely seen and requires special technic for visualization. They are inconstantly present as has been shown by gastroscopic examination.

Changes occurring in the antrum are of great importance to the roentgenologist, because it is in this area that certain direct signs are present which may be the determining factors in making a diagnosis of gastritis. Antral gastritis is of maximum importance since it involves the portion of the stomach which plays a major rôle in gastric motility. In addition, because of alteration in the anatomic form of the antrum and certain changes in peristalsis and the mucosal folds, it may present a confusing picture to the roentgenologist. Gastroscopists have on numerous occasions stated that the mucosal changes of gastritis are detected only by gastroscopy. Golden,[3] however, has called to our attention the fact that there are many changes in gastric form and function that are produced by gastritis, which the roentgenologist must be prepared to recognize.

Golden outlines the rôle of the mucous membrane and muscularis mucosae in the formation of mucosal folds. He describes the movement of the normal mucous membrane of the stomach over the muscle and states that a cephalad movement of the mucosa takes place as antral systole occurs. He believes that in the presence of inflammation this normal movement of the mucous membrane may be hampered or prevented, with consequent failure of the antral folds to flatten under pressure. He also states that in cases of well marked gastritis there are three major disturbances in motility: "1. Peristalsis may be very irregular in depth, and in timing, or may be absent for long periods; the waves seem stiff and fail to develop into antral systoles. 2. Prepyloric narrowing may be present, sometimes relatively slight and amounting merely to failure of the region to expand flexibly to the normal width after a contraction, but sometimes so marked that the antrum appears as a narrow channel. 3. Pylorospasm may be present." As a result of these changes there may be considerable delay in gastric emptying without evidence of organic pyloric narrowing.

The correlation between antral gastritis and hypertrophy of the pylorus is difficult of direct proof, but numerous authors report coincidence of these lesions in resected specimens. Golden believes that at the present time evidence is strongly in favor of the opinion that gastritis is an important factor in the production of hypertrophy of the pyloric muscle. Kirklin and Harris[4] state that the roentgen changes indicating such hypertrophy are (1) lengthening of the pyloric canal and (2) crescentic indentation of the bulbar base. They believe that these two signs will prove to be pathognomonic.

When persistent narrowing of the pyloric antrum is present the radiologist is faced with a dilemma. If he reports the lesion as benign there is always the possibility of malignancy being present, and *vice versa*. It has been stated by Jensen and Rivers[5] that in antral lesions just proximal to the pylorus one cannot be sure from non-operative methods of diagnosis that he is not dealing with malignancy in about 15 per cent of the cases. In a series of 35 ulcerative lesions at and near the pylorus for which resection of the antrum was done, Doub[6] found 20 per cent to be malignant. In 8.5 per cent there was evidence of chronic gastritis and in one case, or 3.0 per cent, syphilis was found.

Golden believes that the most important differential point is the demonstration by pressure methods, or otherwise, of mucosal folds in the narrowed antrum. These should be obliterated by infiltrating carcinoma but occasionally only one wall may be infiltrated, leaving the mucosal folds intact on the other.

It is adequately demonstrated by a review of the literature that the rôle of the roentgenologist in detecting gastritis is a difficult one. The direct roentgen signs which are reliable are few and in many cases difficult to demonstrate. The indirect signs, some of the most valuable of which are found in the antrum, are of great significance and should be carefully studied and evaluated. This is the more important because it is this same area which is fraught with special diagnostic difficulties for every roentgenologist.

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[3] GOLDEN, ROSS: Antral Gastritis and Spasm. J.A.M.A. **109:** 1497–1500, Nov. 6, 1937.

[4] KIRKLIN, B. R., AND HARRIS, M. T.: Hypertrophy of the Pyloric Muscle of Adults: A Distinctive Roentgenologic Sign. Am. J. Roentgenol. **29:** 437–442, April, 1933.

[5] JENSEN, R. M., AND RIVERS, A. B.: Carcinoma or Ulcer Involving the Pyloric Ring: Differential Diagnosis. Proc. Staff Meet., Mayo Clinic **14:** 1–4, Jan. 4, 1939.

[6] DOUB, H. P.: Differential Diagnosis of Pyloric and Prepyloric Ulceration. Am. J. Roentgenol. **43:** 826–831, June, 1940.

ANNOUNCEMENTS AND BOOK REVIEWS

ANNUAL MEETING

RADIOLCGICAL SOCIETY OF NORTH AMERICA

Special attention is called to the forthcoming annual meeting of the Radiological Society of North America to be held at Hotel Fairmont in San Francisco, Dec. 1 to Dec. 5, 1941. In addition to the program, which promises to be rich in scientific interest, the Fourth Annual Refresher Series for Post Graduate Instruction will be presented. The Refresher Series will open on Sunday afternoon, Nov. 30. Further details of the meeting will appear from time to time in these columns.

THE AMERICAN BOARD OF RADIOLOGY

The American Board of Radiology will hold its fall examination in Cincinnati on Sept. 27, 28, and 29. There will be another examination in Atlantic City in June 1942. Those wishing to be examined in Cincinnati should file their applications immediately.

B. R. KIRKLIN, M.D., *Secretary*
Mayo Clinic
Rochester, Minn.

MIDSUMMER RADIOLOGICAL CONFERENCE

The Seventh Midsummer Radiological Conference in the Rocky Mountains will be held at the Hotel Shirley-Savoy, in Denver, Colorado, Thursday, July 31-Saturday, Aug. 2, 1941.

Papers will be presented by Dr. John D. Camp of Rochester, Minn., on "Roentgenologic Observations Concerning Osteoporosis and Its Relation to Systemic Diseases"; by Dr. Atha Thomas of Denver, on "Vascular Tumors of Bone"; by Dr. John T. Murphy of Toledo on "Bone Tumors" and "Carcinoma of the Skin"; by Dr. Wendell G. Scott of St. Louis on "Body Section Radiography as Aid in a Diagnosis of Thoracic Aneurysm or Mediastinal Tumors"; by Dr. Ross Golden of New York City on "Roentgenology of the Small Intestine"; by Dr. D. S. Beilen of Chicago on "Roentgen Therapy in Non-Malignant Conditions"; by Dr. U. V. Portmann of Cleveland on "Irradiation for Diseases of the Thyroid Gland" and "Factors Which Influence the Curability of Malignant Tumors"; and by Dr. Leon J. Menville of New Orleans on "Irradiation Therapy in Endocrine Dysfunction."

On Thursday evening there will be a joint meeting with the Medical Society of the City and County of Denver, Leonard G. Crosby, M.D., presiding. The program will include a symposium on the Gastrointestinal Tract, conducted by Dr. Camp, Dr. Golden, and Dr. Beilen, and a symposium on Irradiation of Malignancy of the Breast, conducted by Dr. Menville and Dr. Portmann, with discussion by Dr. Scott and Dr. Murphy.

AMERICAN CONGRESS OF PHYSICAL THERAPY

The 20th annual scientific and clinical session of the American Congress of Physical Therapy will be held Sept. 1–5, 1941, at The Mayflower, Washington, D. C. For information address the American Congress of Physical Therapy, 30 North Michigan Avenue, Chicago, Ill.

The American Occupational Therapy Association will hold its 25th annual meeting at The Mayflower at the same time. For information concerning this meeting, address Mrs. Meta R. Cobb, 175 Fifth Avenue, New York City. A combined session of the two groups will be held on Wednesday, Sept. 3.

DIRECTORY OF MEDICAL SPECIALISTS

A second edition of the Directory of Medical Specialists has been authorized by the Advisory Board for Medical Specialties, to be ready for distribution in February 1942. This Directory is the official publication of the Advisory Board, and will list the names of approximately 18,000 diplomates of the fifteen American Boards examining candidates for certification in the specialties. Paul Titus, M.D., Pittsburgh, Pennsylvania, of the American Board of Obstetrics and Gynecology, is the Directing Editor.

CLEVELAND RADIOLOGICAL SOCIETY

At a recent meeting of the Cleveland Radiological Society the following officers were elected for the ensuing year: President, H. A. Mahrer, M.D.; Vice-President, E. C. Baker, M.D.; Secretary-Treasurer, J. O. Newton, M.D.

LOUISIANA RADIOLOGICAL SOCIETY

The Louisiana Radiological Society, organized in April 1941, has elected the following officers: President, Stakely F. Hatchette, M.D.; Vice-President, L. A. Fortier, M.D.; Secretary-Treasurer, Johnson R. Anderson, M.D.

MINNESOTA RADIOLOGICAL SOCIETY

The 13th Annual Meeting of the Minnesota Radiological Society was held in Saint Paul, May 27, 1941. Dr. G. T. Nordin of Minneapolis was elected President, Dr. A. U. Desjardins of Rochester, Vice-

President, and Dr. J. P. Medelman of Saint Paul, Secretary. Dr. Leroy Sante of Saint Louis delivered the Annual Carman Lecture before the general session of the Minnesota State Medical Association. His subject was "Roentgen Observations in Chest Injuries."

NEW JERSEY RADIOLOGICAL SOCIETY

At the meeting of the New Jersey Radiological Society held in Atlantic City, May 21, the following officers were elected: President, Dr. N. J. Furst; Vice-President, Dr. W. James Marquis; Secretary, Dr. H. J. Perlberg; Treasurer, Dr. H. A. Vogel; Counselor, Dr. Charles Oderr.

NEW YORK ROENTGEN SOCIETY

The New York Roentgen Society has elected the following officers for the year 1941–42: President, Douglas Quick, M.D.; Vice-President, Eric J. Ryan, M.D.; Secretary, Paul C. Swenson, M.D.; Treasurer, Maurice Pomeranz, M.D.; Member of the Exeeutive Committee, Henry K. Taylor, M.D.

NORTH CAROLINA RADIOLOGICAL SOCIETY

The newly elected officers of the North Carolina Radiological Society are: President, Frederick G. Sigman, M.D.; Vice-President, J. Rush Shull, M.D.; Secretary-Treasurer, Major I. Fleming, M.D.

PITTSBURGH ROENTGEN SOCIETY

Dr. Murray M. Copeland of Baltimore addressed the Pittsburgh Roentgen Society at a dinner meeting in May. Dr. Copeland's subject was The Prognosis of Bone Tumors.

The officers of the Society for the ensuing year are: Dr. Maurice F. Goldsmith, President; Dr. John H. Gemmell, Vice-President; Dr. Harold W. Jacox, Secretary-Treasurer.

TENNESSEE RADIOLOGICAL SOCIETY

At the annual meeting of the Tennessee Radiological Society Dr. Christopher C. McClure was elected President, Dr. Horace D. Gray, Vice-President, and Dr. Franklin B. Bogart, Secretary-Treasurer.

Books Received

BIOLOGIC FUNDAMENTALS OF RADIATION THERAPY. By FRIEDRICH ELLINGER, M.D., Research Fellow, Radiotherapy Department, Montefiore Hospital, New York, N. Y.; Formerly Privatdozent in Radiology at the University of Berlin. A volume of 360 pages with 79 illustrations. Published by Nordeman Publishing Company, Inc., New York, N. Y., 1941. Price: $5.00.

FRACTURES AND OTHER BONE AND JOINT INJURIES. By R. WATSON-JONES, B.Sc., M.Ch.Orth., F.R.C.S., Civilian Consultant in Orthopedic Surgery of the Royal Air Force; Member of War Wounds Committee of Medical Research Council; Member of British Medical Association Committee on Fractures; Member of Council and Chairman of Standing Committee on Fractures, of the British Orthopedic Association; Lecturer in Orthopedic Pathology, Lecturer in Clinical Orthopedic Surgery, and Secretary of the Board of Orthopedic Studies, University of Liverpool; Neurological Surgeon to Special Head and Spinal Centre, Emergency Medical Service; Honorary Orthopedic Surgeon, Royal Liverpool United Hospital (Royal Infirmary); Visiting Surgeon, Robert Jones and Agnes Hunt Orthopedic Hospital; Consulting Orthopedic Surgeon, Royal Lancaster Infirmary, North Wales Sanatorium, Birkenhead, Hoylake, and West Kirby, Wrexham and East Denbighshire, and Garston Hospitals. Second Edition. A volume of 724 pages with 1040 illustrations. Published by The Williams & Wilkins Company, Baltimore, 1941. Price: $13.50.

Book Review

THE STORY OF CLINICAL PULMONARY TUBERCULOSIS. By LAWRASON BROWN, M.D., for many years Director of Trudeau Sanatorium and Lecturer in the Trudeau School of Tuberculosis. A volume of 410 pages, of which 19 pages are devoted to a complete index and 10 pages to bibliography. The bibliography lists 225 American and foreign articles written by 221 authors. Published by The Williams and Wilkins Company, Baltimore, 1941. Price: $2.75.

The author has divided the contents of his book into four parts, starting with an introductory chapter. In it, he has briefly but satisfactorily traced the history of tuberculosis and the development of our knowledge of the disease from earliest antiquity to the present time. He quotes briefly from the works of many physicians but at length from Aretaeus, the Cappadocian, who differentiated empyema from phthisis. All students will welcome the English translation from the works of an ancient master whose description of the far advanced tuberculous patient is thought by the author to surpass that of Hippocrates. Indeed, he believes that it has never been excelled by modern writers.

The remaining three chapters of part one are unique in conception and presentation of their subject matter. The author imagines the visit of a doctor to a patient in the three centuries beginning with the seventeenth. He writes of the several periods, discussing the historical events that occupied the doctor's mind, the part played by the theater, architecture, war, art, and literature in the cultural equipment of the consultant and his pa-

tient. He traces the development, not only of medicine, but also of the sister sciences of chemistry, physics, and hygiene, telling of the discoveries of Newton, the development of the microscope, and the establishment of laboratories and hospitals throughout England and the Continent. He pictures the expanding importance of the physical examination as scientific knowledge advanced, and he unobtrusively introduces biographical sketches of those men whose discoveries became of epochal importance.

The second part of the book is concerned with the beginnings of early diagnosis and a whole chapter is given to a biographical sketch of Laennec and a discussion of the works and writings of his contemporaries.

A chapter on artificial pneumothorax is Dr. Brown's assignment in the third part, and his "story of the stethoscope" is his most interesting contribution in part four.

Students of clinical medicine, especially those interested in the history of medicine, will welcome this book. It is the work of one of the foremost American specialists and teachers of tuberculosis, compiled after his death from the notes he had made in preparation for the lectures which for years he had given to the students of the Trudeau School of Medicine. In collaboration, his friend, Dr. Edward Archibald, has added a chapter on surgical methods of treatment. His associate, Dr. Homer L. Sampson, has written the history of the evaluation of the x-rays and the growth in importance of this method of investigation in the diagnosis of pulmonary tuberculosis. Both Dr. Archibald and Dr. Sampson have followed the general plan adopted by Dr. Brown, so that their chapters are not merely additions, but component parts of the book. The student may study the complete work without the mental adjustments usually needed when reading a book written by more than one authority.

Preserving the Practice of Radiologists Called to Active Military Duty

The following communication was distributed to state councilors of the American College of Radiology and secretaries of all regional radiological societies on April 16:

The following resolution, adopted at the recent meeting of the Board of Chancellors of the American College of Radiology, is transmitted herewith for your attention:

RESOLUTION REGARDING PRACTICE AND POSITIONS OF PHYSICIANS CALLED TO MILITARY SERVICE

"WHEREAS, The national emergency requires the services of many physicians who have been holding hospital and other staff positions; and

"WHEREAS, It is the desire of their colleagues and of all other patriotic citizens to see that the practices and positions of such men are maintained until their return in so far as such is practicable; now therefore be it

"*Resolved*, That the Board of Chancellors of the American College of Radiology request regional radiological societies throughout the country to take all reasonable steps to assist colleagues called to duty and to see that the practices of those colleagues are protected as far as possible either by arranging for consultative service under conditions whereby the identity of the absent radiologist's practice is maintained or by seeing that competent assistants are appointed; and be it further

"*Resolved*, That the Board of Chancellors of the American College of Radiology request the National Hospital Associations to recommend to their component state and regional units that suitable resolutions be adopted, the purport of which shall be that the maintenance of the staff or other appointments of local radiologists should be assured until their return, in good health, from military service; and be it further

"*Resolved*, That the hospital physician called to duty shall, if possible, be given the opportunity to select his substitute, the appointment of whom shall be subject to the approval of the hospital authorities and staff."

In connection with hospital appointments, we call your attention to the following resolutions adopted by the Board of Trustees of the American Hospital Association on February 15, 1941:

"*Resolved*, That the Board of Trustees recommend to member hospitals and other hospitals that, if consistent with local conditions, leaves of absence be granted to all employees during the period of their active military service."

While directors of hospital radiological departments are not employees of the hospital, and while the Board of Chancellors does not like to consider the appointments of staff radiologists in the same connection with employed personnel of the hospital, it is nevertheless true that the staff appointment of a radiologist presents a somewhat different problem than the positions of other physicians on the staff. With this point in mind, the Executive Committee of the Board of Chancellors conferred with the Council on Administrative Practice of the American Hospital Association during the Chancellors' meeting in February. Our Committee was assured by the Council that the resolution quoted above was meant to include the staff appointments of directors of hospital radiological departments.

The Board of Chancellors suggests that regional radiological societies consider the preparation of an agreed model contract which could be used by a member of their local society in obtaining a *locum tenens* for his hospital position or private office. The suggested contract should probably provide for an agreed percentage of the gross income from the practice to be retained by the *locum tenens*, the balance over necessary expenses to be placed in trust for the absentee. It is further suggested that the model contract contain a negative covenant by which the *locum tenens* agrees not to enter the practice of radiology in the community for a reasonable length of time after the absent partner returns to civil practice and the contract is terminated. The Board of Chancellors feels that, in so far as local conditions permit, each hospital radiologist who is called to service should be given the privilege of selecting his substitute or *locum tenens* and making the contract with the latter himself, instead of having the agreement made between the hospital superintendent and the *locum tenens*.

MAC F. CAHAL
Executive Secretary

Radiological Societies in North America

Editor's Note.—Will secretaries of societies please cooperate with the Editor by supplying information to keep these notices accurate and up to date? Please send information to Howard P. Doub, M.D., Henry Ford Hospital, Detroit, Mich.

UNITED STATES

CALIFORNIA

California Medical Association, Section on Radiology.—Secretary, Wilbur Bailey, M.D., 2007 Wilshire Blvd., Los Angeles.

Los Angeles County Medical Association, Radiological Section.—Secretary, Wilbur Bailey, M.D., 2007 Wilshire Blvd.; Meets second Wednesday of each month at County Society Building.

Pacific Roentgen Society.—Secretary-Treasurer, L. Henry Garland, M.D., 450 Sutter St., San Francisco. Society meets annually during annual meeting of the California Medical Association.

San Francisco Radiological Society.—Secretary, J. Maurice Robinson, M.D., University of California Hospital. Meets monthly on third Thursday at 7:45 P.M., for the first six months at Toland Hall (University of California Medical School) and for the second six months at Lane Hall (Stanford University School of Medicine).

COLORADO

Denver Radiological Club.—Secretary, Paul R. Weeks, M.D., 520 Republic Bldg. Meets third Friday of each month at homes of members.

CONNECTICUT

Connecticut State Medical Society, Section on Radiology.—Secretary-Treasurer, Max Climan, M.D., 242 Trumbull St., Hartford. Meetings bimonthly, on second Thursday. Place of meeting selected by Secretary.

FLORIDA

Florida Radiological Society.—Secretary-Treasurer, Elliott M. Hendricks, M.D., 314 Sweet Bldg., Fort Lauderdale. The next meeting will be at the time of the annual meeting of the Medical Association of Florida in the spring.

GEORGIA

Georgia Radiological Society.—Secretary-Treasurer, Robert C. Pendergrass, M.D., Prather Clinic Bldg., Americus. Meetings twice annually, in November and at the annual meeting of the Medical Association of Georgia in the spring.

ILLINOIS

Chicago Roentgen Society.—Secretary, Chester J. Challenger, M.D., 3117 Logan Blvd. The Society meets at the Palmer House on the second Thursday of October, November, January, February, March, and April.

Illinois Radiological Society.—Secretary-Treasurer, William DeHollander, M.D., St. Johns' Hospital, Springfield. Meetings quarterly by announcement.

Illinois State Medical Society, Section on Radiology.—Secretary, Earl E. Barth, M.D., 303 E. Chicago Ave., Chicago.

INDIANA

The Indiana Roentgen Society.—Secretary-Treasurer, Clifford C. Taylor, M.D., 23 E. Ohio St., Indianapolis. Annual meeting in May.

IOWA

The Iowa X-ray Club.—Holds luncheon and business meeting during annual session of Iowa State Medical Society.

KENTUCKY

Kentucky Radiological Society.—Secretary-Treasurer, Joseph C. Bell, M.D., 402 Heyburn Bldg., Louisville. Meeting annually in Louisville, third Sunday afternoon in April.

LOUISIANA

Louisiana Radiological Society.—Secretary-Treasurer, Johnson R. Anderson, M.D., North Louisiana Sanitarium, Shreveport. Meets annually at same time as State Medical Society. Next meeting, New Orleans, April 1942.

Shreveport Radiological Club.—Secretary-Treasurer, W. R. Harwell, M.D. Meetings monthly on the second Wednesday, at the offices of the various members.

MARYLAND

Baltimore City Medical Society, Radiological Section.—Secretary, Walter L. Kilby, M.D., 101 W. Read St. Meetings are held the third Tuesday of each month.

MICHIGAN

Detroit X-ray and Radium Society.—Secretary-Treasurer, E. R. Witwer, M.D., Harper Hospital, Detroit. Meetings first Thursday of each month from October to May, inclusive, at Wayne County Medical Society club rooms, 4421 Woodward Ave., Detroit.

Michigan Association of Roentgenologists.—Secretary-Treasurer, J. E. Lofstrom, M.D., 1536 David Whitney Bldg., Detroit. Meetings quarterly by announcement.

MINNESOTA

Minnesota Radiological Society.—Secretary, John P. Medelman, M.D., 572 Lowry Medical Arts Bldg., St. Paul. Meetings quarterly.

MISSOURI

The Kansas City Radiological Society.—Secretary, P. E. Hiebert, M.D., 907 North Seventh St. (Huron Bldg.), Kansas City, Kansas. Meetings last Thursday of each month.

The St. Louis Society of Radiologists.—Secretary, Wilbur K. Mueller, M.D., University Club Bldg. Meets on fourth Wednesday of October, January, March, and May, at a place designated by the president.

NEBRASKA

Nebraska Radiological Society.—Secretary, D. A. Dowell, M.D., 816 Medical Arts Bldg., Omaha. Meetings third Wednesday of each month at 6 P.M. in either Omaha or Lincoln.

NEW ENGLAND

New England Roentgen Ray Society.—(Maine, New Hampshire, Vermont, Massachusetts, and Rhode Island.) *Secretary,* Hugh F. Hare, M.D., Lahey Clinic, Boston, Mass. Meets monthly on third Friday at Boston Medical Library.

NEW JERSEY

Radiological Society of New Jersey.—Secretary, H. J. Perlberg, M.D., Trust Co. of New Jersey Bldg., Jersey City. Meetings at Atlantic City at time of State Medical Society and midwinter in Newark as called by president.

NEW YORK

Associated Radiologists of New York, Inc.—Secretary, William J. Francis, M.D., 210 Fifth Ave., New York City. Regular meetings the first Monday evening of the month in March, May, October, and December.

Brooklyn Roentgen Ray Society.—Secretary-Treasurer, L. J. Taormina, M.D., 1093 Gates Ave. Meetings held the fourth Tuesday of every month, October to April.

Buffalo Radiological Society.—Secretary-Treasurer, Joseph S. Gianfranceschi, M.D., 610 Niagara St. Meetings second Monday evening each month, October to May, inclusive.

Central New York Roentgen Ray Society.—Secretary-Treasurer, Carlton F. Potter, M.D., 425 Waverly Ave., Syracuse. Meetings are held in January, May, and October, as called by Executive Committee.

Long Island Radiological Society.—Secretary, Marcus Wiener, M.D., 1430 48th St., Brooklyn. Meetings fourth Thursday evening each month at Kings County Medical Bldg.

New York Roentgen Society.—Secretary, Paul C. Swenson, M.D., Presbyterian Hospital, New York, N. Y.

Rochester Roentgen-ray Society.—Secretary, S. C. Davidson, M.D., 277 Alexander St. Meetings at convenience of committee.

NORTH CAROLINA

Radiological Society of North Carolina.—Secretary-Treasurer, Major I. Fleming, M.D., 404 Falls Road, Rocky Mount. Meeting with State meeting in May, and meeting in October.

OHIO

Ohio Radiological Society.—Secretary, J. E. McCarthy, M.D., Cincinnati. The next meeting will be held at the time and place of the annual meeting of the Ohio State Medical Association.

Cleveland Radiological Society. — Secretary-Treasurer, J. O. Newton, M.D., 13921 Terrace Road, East Cleveland. Meetings at 6:30 P.M. at the Mid-day Club, in the Union Commerce Bldg., on fourth Monday of each month from October to April, inclusive.

Radiological Society of the Academy of Medicine (Cincinnati Roentgenologists).—*Secretary-Treasurer,* Justin E. McCarthy, M.D., 707 Race St. Meetings held third Tuesday of each month.

PENNSYLVANIA

Pennsylvania Radiological Society.—Secretary-Treasurer, L. E. Wurster, M.D., 416 Pine St., Williamsport; The Society meets annually; time and place of next meeting will be announced later.

The Philadelphia Roentgen Ray Society.—Secretary, Barton R. Young, M.D., Temple University Hospital, Philadelphia. Meetings held first Thursday of each month at 8:15 P.M., from October to May, in Thomson Hall, College of Physicians, 21 S. 22nd St., Philadelphia.

The Pittsburgh Roentgen Society.—Secretary-Treasurer, Harold W. Jacox, M.D., 4800 Friendship Ave., Pittsburgh, Pa. Meetings are held on the second Wednesday of each month at 4:30 P.M., from October to June, at the Pittsburgh Academy of Medicine, 322 N. Craig St.

SOUTH CAROLINA

South Carolina X-ray Society.—Secretary-Treasurer, Malcolm Mosteller, M.D., Columbia Hospital, Columbia. Meetings in Charleston on first Thursday in November, also at time and place of South Carolina State Medical Association.

TENNESSEE

Memphis Roentgen Club.—Chairmanship rotates monthly in alphabetical order. Meetings second Tuesday of each month at University Center.

Tennessee Radiological Society.—Secretary-Treasurer, Franklin B. Bogart, M.D., 311 Medical Arts Bldg., Chattanooga. Meeting annually with State Medical Society in April.

TEXAS

Texas Radiological Society.—Secretary-Treasurer, L. W. Baird, M.D., Scott and White Hospital, Temple. Meets annually.

VIRGINIA

Virginia Radiological Society.—Secretary, Charles H. Peterson, M.D., 603 Medical Arts Bldg., Roanoke.

WASHINGTON

Washington State Radiological Society.—Secretary-Treasurer, Kenneth J. Holtz, M.D., American Bank Bldg., Seattle. Meetings fourth Monday of each month at College Club, Seattle.

WISCONSIN

Milwaukee, Roentgen Ray Society.—Secretary-Treasurer, Irving I. Cowan, M.D., Mount Sinai Hospital, Milwaukee. Meets monthly on first Friday at the University Club.

Radiological Section of the Wisconsin State Medical Society.—Secretary, Russel F. Wilson, M.D., Beloit Municipal Hospital, Beloit. Two-day annual meeting in May and one day in connection with annual meeting of State Medical Society, in September.

University of Wisconsin Radiological Conference.—Secretary, E. A. Pohle, M.D., 1300 University Ave., Madison, Wis. Meets every Thursday from 4 to 5 P.M., Room 301, Service Memorial Institute.

CANADA

Section on Radiology, Canadian Medical Association.—Secretary, W. J. Cryderman, M.D., Medical Arts Bldg., Toronto.

Section on Radiology, Ontario Medical Association.—Secretary, W. J. Cryderman, M.D., 474 Glenlake Avenue, Toronto.

Canadian Association of Radiologists.—Honorary Secretary-Treasurer, A. C. Singleton, M.D., Toronto.

La Société Canadienne-Française d'Eléctrologie et de Radiologie Médicales.—General Secretary, Origène Dufresne, M.D., Institut du Radium, Montreal. Meetings are held the third Saturday of each month, generally at the Radium Institute, 4120 East Ontario Street, Montreal; sometimes, at homes of members.

CUBA

Sociedad de Radiología y Fisioterapia de Cuba.—Offices in Hospital Mercedes, Havana. Meetings are held monthly.

ABSTRACTS OF CURRENT LITERATURE

ROENTGEN DIAGNOSIS

The Head and Neck

The Chest

The Digestive Tract

The Spleen

The Skeletal System

RADIOTHERAPY

Malignant Conditions

Non-Malignant Conditions

Dosage and Technic

MISCELLANEOUS

ROENTGEN DIAGNOSIS

THE HEAD AND NECK

Intracranial Tumors: Study of 467 Histologically Verified Cases. A. B. Baker. Minnesota Med. **23:** 696–703, October 1940.

This classification is based on 467 brain tumors studied at the University of Minnesota.

Tumors of nerves comprised only 1.5 per cent of the cases. They include acoustic neuromas, optic nerve gliomas, and cerebral neurofibromatosis.

Tumors of the meninges made up 18.4 per cent of the series. These are, as a rule, benign encapsulated masses that rarely invade the brain but do compress it. They frequently invade the skull, producing increased vascularization, hyperostosis, or erosion. These tumors are sometimes called arachnoidal fibroblastomas.

Gliomas occurred as follows: astrocytoma, 45 per cent; astroblastoma, 5 per cent; spongioblastoma polare, 2.9 per cent; glioblastoma multiforme, 4 per cent; medulloblastoma, 7.5 per cent; oligodendroglioma, 2.1 per cent; pinealoma, 2.1 per cent; ependymoma, 7.1 per cent; neuroepithelioma, 1.4 per cent; papilloma of choroid plexus 0.4 per cent; undetermined 0.7 per cent.

Vascular tumors are grouped as angiomas and angioblastomas; they comprised 2.7 per cent of all the intracranial tumors.

Tumors of mixed tissue include dermoids and teratomas. Only one such tumor was included in the group.

Hypophyseal tumors: Both pituitary adenomas and craniopharyngiomas have been placed in this group. The pituitary adenomas are grouped as chromophobe, acidophil, and basophil. The latter two are usually small in size and often escape post-mortem detection. The chromophobe tumors are larger, and may distort the chiasm. The adenomas made up 4.6 per cent of the group. The craniopharyngiomas are subdivided into Rathke pouch tumors and adamantinomas.

Granulomata made up 6 per cent of all the intracranial growths. This group includes tuberculomas and gummas.

The incidence of the various groups is correlated with the age and sex of the patients, and compares with similar classifications from the larger clinics.

PERCY J. DELANO, M.D.

Osteomyelitis of the Skull Following Frontal Sinusitis. A. S. Macmillan. J.A.M.A. **115:** 1176–1178, Oct. 5, 1940.

The author considers first the roentgenologic aspects of acute and chronic sinus infection. The minimum number of views that should be taken to arrive at an intelligent interpretation of the density of a sinus is four, namely, a lateral, a postero-anterior, a Waters position in the prone, and a fluid-level position. The lateral view must be examined for the depth of the sinus and for the thickness of the anterior and posterior walls. The thickness of the cortex of the skull relative to that of the medulla must be noted. When the nasofrontal duct becomes completely or partially blocked, the membrane thickened, and its secretion retained within the sinus, there is surprisingly little increase in density over the normal. Often the difference in density between the two frontal sinuses is so slight, even when they are completely filled, that other means of diagnosis must be sought. One must watch for the decalcification of the bone that results from acute inflammation of the mucous membrane. This occurs in from seven to ten days after onset of infection of the mucous membrane. In subacute and chronic sinusitis, lasting a matter of weeks, there is an increase in density of the anterior and posterior walls and the surrounding bone, nature's walling off process. This eventually results in sclerosis, so consistently seen in chronic infections.

The progress of infection in acute frontal sinusitis is reflected in the change in density, loss of outline, and reaction in the surrounding bone. Minute perforations can be detected in the anterior views and are one of the early signs of bone infection, but infection must be present a week before these signs become evident. Involvement of the posterior wall of the frontal sinus may be determined by noting the change in the pattern of the composite picture of the anterior and posterior walls. In the presence of clinical evidence, however, one should not wait for roentgenologic signs of the disease.

C. G. SUTHERLAND, M.D.

Osteomyelitis of the Frontal Bone as a Complication of Frontal Sinusitis. H. P. Mosher. J.A.M.A. **115:** 1179–1182, Oct. 5, 1940.

Osteomyelitis of the frontal bone is of two types—the fulminating, rapidly spreading form, of which swimming is a frequent cause, and the less virulent localizing form which tends to burn itself out and form sequestra. If the patient is septic, with pitting edema of the skin of the forehead half way to the hair line, or at the hair line, as oftens happens, the author classes the case as of the fulminating type.

The diploetic veins of the skull are numerous and connect superficially with the veins of the scalp and deeply with the blood sinuses of the skull. The diploetic veins of the frontal bone are a continuation of the veins of the mucous membrane of the frontal sinus. Relatively they are very large and are absent from the frontal bone in only 3 per cent of cases. The infection in the frontal sinus produces thrombophlebitis of the veins of the mucous membrane and of the diploetic veins of the frontal bone. The infection spreads to the bone marrow in which the veins are located. The branches of the diploetic veins run outward to the periosteum of the frontal bone and inward to the dura. The dura seems to attract the infection more strongly than the periosteum.

Pitting edema of the skin of the forehead indicates involvement of the frontal bone. In fulminating cases this rapidly extends to the hair line. X-ray examination shows a cloudy frontal sinus and gives the number and position of the diploetic veins. It is not positive for osteomyelitis until seven to ten days after the onset of pitting edema.

The treatment of choice is removal of practically the entire frontal bone. C. G. SUTHERLAND, M.D.

Roentgen Signs of Mastoiditis and Its Complications. Vincent C. Johnson. J.A.M.A. **115:** 510–513, Aug. 17, 1940.

Four projections were selected on the basis of experience for employment in routine mastoid examination. Beginning with the standard oblique lateral projection of Law, there have been added the oblique postero-anterior projection described by Stenvers, the fronto-occipital projection of Towne and, more recently, an axial projection of the base of the skull in the mentovertical direction. To enhance further the diagnostic value of the roentgenograms thus obtained, stereoscopic technic was employed throughout, and in all cases both temporal bones were examined.

The roentgenologic signs of diseased mastoid cells are well known. Early suppuration results in simple clouding, to be followed in more advanced stages by

recognizable absorption of the cell walls. If untreated, the process may progress to the formation of large bone abscesses. If the infection subsides, varying degrees of bone regeneration will occur during repair, causing increased thickness and density of cell walls. More advanced stages of suppurative disease usually require surgical exenteration of diseased cells and the establishment of drainage. The roentgen examination may be of further assistance, when it is necessary to plan operative methods of treatment, by demonstrating the position and caliber of the sigmoid sinus. The roentgenologist can and should describe the position of this sinus in every report. It is wise to reexamine the mastoid after surgical intervention to provide graphic evidence regarding the completeness of the operation and the location and status of surgically inaccessible cells which may come under suspicion at a later date.

Complications closely localized to the temporal bone and recognizable directly on roentgen examination are relatively few. Microscopic evidence of cholesteatoma has been reported as occurring in 50 per cent of cases coming to operation (Macmillan: Am. J. Roentgenol. **36:** 747, 1936). Bone destruction surrounding cholesteatoma was recognized preoperatively with the use of the Law projection in 45 per cent of the cases examined, while with the additional aid of the occipital projection the percentage of preoperative diagnosis of cholesteatoma was increased to 68 per cent. The roentgen signs of the bone destruction produced by cholesteatoma are usually distinct. Characteristically the bone defect is smoothly rounded and rimmed with a wall of sclerotic bone. Destruction of this type is most commonly encountered in the mastoid antrum or extending laterally from this region.

Roentgen signs of suppurative disease involving cells within the petrous portion of the temporal bone (petrositis means inflammatory disease of that part which lies medial and anterior to the arcuate eminence and the superior semicircular canal) do not vary from those which are useful in estimating the status of the mastoid process, although signs of clouding and cell wall destruction are as a rule recognized with greater difficulty. Roentgen signs of petrositis can be considered of value only when they confirm clinical manifestations of the disease or when they are helpful in determining the most desirable method of surgical treatment.

Other complications mentioned are brain abscess, cranial osteomyelitis, and primary tumors, the last two being of rare occurrence. C. G. SUTHERLAND, M.D.

Internal Frontal Hyperostosis. P. H. Rossier and J. P. Secretan. Schweiz. med. Wchnschr. **70:** 994–1004, Oct. 19, 1940.

The authors present the results of a study of 39 patients with internal frontal hyperostoses. They show the interdependence of the various cranial thickenings and adopt Moore's classification (see Arch. Neurol. & Psychiat. **35:** 975, 1936). The condition leads to a typical clinical syndrome, which is so well characterized that the diagnosis may be suspected solely on clinical grounds, although radiographic confirmation is needed. The picture is that of an infundibulo-hypophyseal defect, with changes in the glucose tolerance curve and the specific dynamic action of proteins, adiposity, amenorrhea, sleep disturbances, and hypercholesteremia. The phenomenon is not limited to the aged, often beginning at adolescence without more severe endocrine disturbances than are observed when it occurs later. Treatment with hypophyseal extracts seems to have given good results.

LEWIS G. JACOBS, M.D.

THE CHEST

Early Diagnosis of Pulmonary Tuberculosis. Manuel Quisumbing. Dis. of Chest **6:** 240–242, August 1940.

The author emphasizes the necessity of periodic examinations of all persons exposed to tuberculosis and recommends a systematic investigation or physical examination of groups of school children, teachers, factory laborers, employees, etc., in order that an early diagnosis may be made. Since the clinician must first suspect the disease in order that diagnosis be attempted, the excellent recapitulation of symptoms and signs suggesting tuberculosis is the greatest contribution made by this article.

Such symptoms are persistent hoarseness, obstinate cough or tracheal pharyngitis, frequent and protracted colds (tuberculous bronchitis), anorexia, sensation of oppression in the epigastrium, nausea, sensation of fullness in the stomach, constipation and diarrhea, afternoon fever, occasional night sweats, lassitude, general weakness, a disparity between fever and its symptoms, dry pleurisy, pleurisy with effusion, intercostal pain or backache especially when associated with pyrexia not responding to antipyretics, hemoptysis, diminution or absence of menstrual flow in young women, reflex pain, intercostal pain, pains in the side between the axillary lines, pains in the abdomen which may simulate a gastric or hepatic lesion or appendicitis, pains that may be attributed to a latent cortico-pleuritis.

WM. H. GILLENTINE, M.D.

Early Diagnosis of Pulmonary Tuberculosis in General Practice. R. H. Stiehm. Wisconsin M.J. **39:** 831–834, October 1940.

Since minimal and even moderately advanced pulmonary tuberculosis can be present without producing symptoms or physical signs, early diagnosis is dependent on repeated roentgen examination of infected persons as determined by the Mantoux test. The small size of an infiltrate or absence of change in serial roentgenograms does not prove that the lesion is either benign or inactive. Guinea-pig inoculation with the morning fasting gastric contents is the most delicate method for determining the activity of a tuberculous lesion. The erythrocyte sedimentation rate is usually normal in the active minimal lesion. In trained hands roentgenoscopic examination is a valuable addition to films in detecting early tuberculosis and in following its progress.

LESTER W. PAUL, M.D.

Serial Fluoroscopy in the Army. O. Spühler. Schweiz. med. Wchnschr. **70:** 653–654, July 6, 1940.

Fluoroscopy of the chest was first employed in the Swiss army in 1921. Although experience has shown that it is somewhat inferior to film methods of examination, it has raised the number of detected cases of early tuberculosis from about 1 per cent of those examined by clinical methods only, to between 2 and 3 per cent.

The procedure has great public-health value, as it singles out undetected cases, that is those without symptoms, and as a by-product picks up cases of heart disease which are often missed clinically. Many civil benefits, such as life insurance and health insurance, are affected as well as military service. The cost is negligible compared with the advantages.

Some incomplete statistics are given as to the diseases discovered. In 675 patients, there were 4 examples of open tuberculosis, 1 small pleural effusion, 14 shadows considered "non-specific," 1 spontaneous pneumothorax without evidence of tuberculosis. In another group, of 595 cases, there were 10 examples of open tuberculosis, 2 small pleural effusions, 1 instance of bilaterally neoplastic hilar glands.

While this article demonstrates that serial fluoroscopic examination of the chest is of real value, it fails to convince one that, where possible, one or another type of film examination should not be made in preference. LEWIS G. JACOBS, M.D.

Tuberculosis Case Findings through Mass Surveys in Adult Groups. Abraham E. Jaffin. Dis. of Chest **6:** 168–169, June 1940.

Mass surveys of industrial workers, wholesale and retail business houses, W.P.A. workers, recipients of public relief, food handlers, transportation employees, inmates of custodial institutions, police and fire departments, made during the past few years have led to the discovery of the disease in 2.3 to 5.2 per cent of those examined, the number increasing with age. Such studies are desirable since they disclose the disease in an early form, thereby preventing spread and increasing the value of treatment. As the author states, tuberculosis will become less a problem when the doctors have more patients and fewer hopeless cases, and the industrial hazard will be less when the people have more health and less lingering illness.

WM. H. GILLENTINE, M.D.

Tuberculosis among Nurses. Paul V. Davis. Dis. of Chest **6:** 214–218, July 1940.

The author summarizes various studies made of the incidence of tuberculosis in nurses since this problem was brought to the attention of the profession by Heimbeck, in Oslo, Norway, in 1927 (see Arch. Int. Med. **41:** 336, 1928). The fact is stressed that the greatest single factor in the spread of the disease is contact. Tuberculin surveys and observations of positive and negative reactions, with routine chest plates from time to time, are recommended as a means of insuring early treatment. WM. H. GILLENTINE, M.D.

Tuberculosis Case-Finding in University of Kansas Students. R. I. Canuteson. J. Kansas M. Soc. **41:** 417–420, October 1940.

The author describes the procedure followed in the tuberculous case-finding program at the University of Kansas. Students reacting positively to the tuberculin test are examined roentgenologically. Those with definite or suspicious findings are investigated thoroughly by clinical and laboratory methods. Among the 9,414 students tuberculin tested in the past eight years there have been 33 cases of the reinfection type of tuberculosis, an incidence of 0.35 per cent. Thirteen of these have been proved to be active. During the same period, 12 additional cases of tuberculosis were diagnosed among students who had not been previously tested. LESTER W. PAUL, M.D.

Miniature X-Ray Film in the "Total Survey." Frederick Tice. J.A.M.A. **115:** 1254–1257, Oct. 12, 1940.

The author reports a mass or "total" x-ray survey carried out in a selected district in Chicago known to have a high tuberculosis mortality. The photoroentgenographic unit developed by one of the equipment houses in collaboration with Potter was used throughout. The size of the film was 4 × 5 inches (10 × 12.7 cm.) and the approximate cost of each film was 5 cents. The apparatus was installed in a mobile x-ray unit, complete with dressing rooms, ventilator, and fans, mounted on a balloon-tired 2½-ton truck and supplied with 200 feet of cable for connection with the power outlets.

X-ray examination within easy walking distance was the prime objective. Contemporaneous with the campaign of education and publicity, ten field nurses were assigned to make a house-to-house canvass of the one-half square mile immediately surrounding the temporary station established. Ten thousand persons were examined in sixty-six days.

The fluorograms were examined by a committee of four physicians, all experienced in x-ray interpretation. Opinion had to be unanimous on every diagnosed case. If one member of the committee disagreed, the patient was classed as a "suspect" and a roentgenogram on a 14 × 17 inch (35.7 × 43.3 cm.) film was ordered. Fluorograms showing suspicious or abnormal markings were similarly followed up.

The fluorograms were studied in magnification to 8 × 10 inches (20.4 × 25.4 cm.) in an especially constructed viewing apparatus.

Of the 10,000 persons on whom fluorographic examinations were made, 9,348 were fully analyzed and charted. Of these, 8,942 were Negroes, 340 white persons, 34 Mexicans, and 32 others. A total of 351 (3.75 per cent) cases of the reinfection type of pulmonary tuberculosis were elicited. The findings were negative in 8,317 (88.98 per cent), of those examined, and 680 were classed as suspects. Of the 680 suspects, 287 had been re-examined with 14 × 17 inch film at the time of the report; 45 or 15.7 per cent were found to have tuberculosis, 108 or 37.6 per cent were negative, while the remainder were still regarded as suspects. A higher incidence of pulmonary tuberculosis in white males and females agreed with the findings of a similar survey made in the Harlem district of New York City.

Tice believes that the fluorogram represents an efficient public health instrument destined probably to general acceptance by all communities with a serious tuberculosis problem. He is convinced that future developments lie in the direction of still smaller and cheaper films, probably 35-mm. film. The technical difficulties involved are not insurmountable and the reduction in cost, reaching as low as a cent per film, constitutes a perennial inducement to further research.

C. G. SUTHERLAND, M.D.

X-Ray Interpretation in Tuberculous Case-Finding. G. M. Tice. J. Kansas M. Soc. **41:** 420–422, October, 1940.

The necessity for diligence and care in the interpretation of chest films in tuberculosis case-finding surveys is stressed. It is emphasized that not all upper lobe infiltrates are tuberculous, although such usually is the case. In early tuberculosis, the radiologist frequently can give no opinion concerning the activity of the lesion. This must be determined by progress studies and by other laboratory procedures. Roentgenograms of good technical quality are essential and fluoroscopic examination has a place as an adjunct to the roentgenographic study. With adequate roentgen examination the early case of tuberculosis can usually be diagnosed before it can be recognized clinically. LESTER W. PAUL, M.D.

Tracheobronchial Complications of Pulmonary Tuberculosis. R. C. Carpenter. J. Iowa M. Soc. **30:** 440–445, September 1940.

Within recent years involvement of the trachea and bronchi in pulmonary tuberculosis has been recognized with increasing frequency. Thus among 93 selected cases, the author found bronchoscopic evidence of such involvement in 18 per cent. The condition may be suspected both clinically and roentgenologically. The following roentgen signs are listed as being suggestive of tracheobronchial tuberculosis: (1) continuous or intermittent atelectasis of one or more lobes; (2) failure of a cavity to collapse after adequate collapse

therapy; (3) failure of a lung to re-expand after pneumothorax; (4) "tennis-ball" cavity; (5) cavities with fluid levels; (6) narrowing of the trachea or bronchus demonstrable either with or without iodized oil; (7) cavities with extensive perifocal reaction; (8) hilar cavities or hilar infiltrations; (9) cavities which appear or disappear or vary in size; (10) transient areas of infiltration in the lung parenchyma; (11) recent spread of the tuberculosis. Clinically, an asthmatoid wheeze and hemoptysis occurring with quiescent pulmonary tuberculosis are the two most important findings. The diagnosis can be established by bronchoscopy, although the latter is of little value in treatment. No deleterious effects from bronchoscopy have been noted in patients with active pulmonary tuberculosis. LESTER W. PAUL, M.D.

Studies in Asbestosis. Moses J. Stone. Dis. of Chest **6:** 170–171, June 1940.

This is a condensation of a longer article in Am. Rev. Tuberculosis (**41:** 12–21, January 1940). One hundred and eighty patients employed in a factory manufacturing asbestos brake lining, with an average exposure time to asbestos dust of five to fifteen years, were examined. Thirty-two showed no evidence of disease. Seventy-eight showed a definite limitation of chest expansion with some roentgen evidence of increased lung markings. Fifty-four had definite symptoms and x-ray evidence of pulmonary fibrosis. Sixteen had marked pulmonary involvement. Eight to eleven years were necessary for the development of changes. Dyspnea, cough, fatigue, expectoration, tightness of the chest, anorexia, and loss of weight occurred. Physical findings were meager. Dullness at the lung bases with hyperresonance at the apices, diminished chest expansion, prolonged expirations with some high-pitched dry râles were found.

The roentgen findings are, in the early stage, slight increase in density in the lung bases; in the later stages, thickening of the pleura, a fine lace-like fibrosis, a typical granular or "ground-glass" appearance in the mid-lung with nodular areas, increased bronchovascular markings, pericardial and pleural thickening, elevation of the diaphragm. The x-ray appearance was sometimes altered by congestion due to associated heart disease, which apparently causes the greater part of the dyspnea of effort early in the course. Bronchiectasis, bronchitis, and bronchopneumonia were more frequently associated with asbestosis than with pulmonary tuberculosis. The condition continues to progress even after exposure ceases.

WM. H. GILLENTINE, M.D.

Clinical Aspect and Incidence of Spontaneous Pneumothorax in 49,198 Fluoroscopic Examinations. Cirilo Santos and F. Tanchanco, Jr. Bull. Quezon Inst. **1:** 213–222, July 1940.

Fifty-seven cases of spontaneous pneumothorax were discovered in nearly 50,000 routine fluoroscopic and radiologic examinations of dispensary cases between 1938 and 1940, an incidence of 0.11 per cent. Ninety-five per cent of this group were due to pulmonary tuberculosis. Five per cent occurred in persons having no demonstrable pulmonary disease. The right lung was affected most frequently. The male–female ratio of incidence was four to two.

WM. H. GILLENTINE, M.D.

Postoperative Atelectasis: Anesthetist's Part in Diagnosis and Treatment. Lloyd H. Mousel. J.A.-M.A. **115:** 899–902, Sept. 14, 1940.

It has been suggested that edema of the mucous membranes, similar to angioneurotic edema, might produce enough obstruction to cause pulmonary collapse. Bronchial spasm has also been suggested as an etiologic factor, but this hypothesis seems unlikely, since bronchial spasm usually produces the antithetic condition; that is, emphysema with bilateral distribution. According to the "vasomotor theory" dilatation and stasis in the blood vessels may produce obstruction in the bronchioles by an outpouring of secretion. Most instances of postoperative atelectasis are caused, in the author's opinion, by mechanical obstruction; that is, actual plugging of a bronchus or several bronchi by tenacious secretions which have collected in the tracheobroncial tree during anesthesia, by mucopurulent material which was present preoperatively, or by mucus, blood, or vomitus aspirated into the trachea either during or immediately following anesthesia.

The onset of actual atelectasis probably is gradual. The bronchus becomes plugged, causing preliminary emphysema. If this plug is not removed by aspiration or if the patient is unable to remove the plug by coughing or by change in position, the air in the involved region is slowly absorbed in the blood stream until pulmonary collapse is complete.

If atelectasis does occur postoperatively, bronchial obstruction should be removed as soon as the condition has been recognized, for if it is allowed to exist for many hours secondary pneumonitis or pulmonary suppuration is likely to develop.

C. G. SUTHERLAND, M.D.

Congenital Solitary Cyst of the Lung. Giulio Calchi-Novati. Radiol. med. **27:** 556–576, July 1940.

This is a lengthy, well illustrated essay on congenital solitary cysts of the lung. The literature is reviewed and an excellent clinical study is given. The value of x-ray in the differential diagnosis is stressed.

A. MAYORAL, M.D.

Examination of the Heart and Lungs by the Cardiocairographic Method. I. Seth Hirsch. Am. Heart J. **20:** 195–204, August 1940.

Using an apparatus constructed by Myron Schwartzschild, which synchronizes the roentgen exposure with the action currents of the heart, the author has studied heart shadows in maximum systole and maximum diastole. He believes that the method will contribute to more accurate heart measurements, providing the maximum degree of sharpness and detail of the pulmonic margins by avoiding blurring of hilum structures due to heart movement. With other technics blurring is unavoidable even with exposures reduced to 1/30 of a second. The author also feels that more perfect visual fusion may be obtained in stereoscopic roentgenograms if maximum sharpness and detail are preserved by this device. WM. H. GILLENTINE, M.D.

Diagnosis of Coronary Occlusion and Myocardial Infarction by Fluoroscopic Examination. Arthur M. Master, Richard Gubner, Simon Dach, and Harry L. Jaffe. Am. Heart J. **20:** 475–485, October 1940.

Fluoroscopic examination of 80 patients with cardiac infarction revealed pulsation abnormalities in 73 per cent. Reversal of pulsation, that is systolic expansion, was found in 29 cases; partial reversal appearing as a lag in 11 cases, absence of pulsation in 2 cases, and a diminution of pulsation in 17 cases. No such abnormalities were found in a control group of normal hearts. These observations were checked against roentgenkymograms taken immediately after the fluoroscopic examination. Reversal of pulsation is also seen in enlarged hearts without infarction. Pulsation abnormalities may help to evaluate the degree of myocardial involvement after recovery from an acute attack. WM. H. GILLENTINE, M.D.

Roentgenkymography of the Heart: Its Clinical Applications and Limitations. Richard Gubner, J. Hamilton Crawford, Wilbur A. Smith, and Harry E. Ungerleider. Am. Heart J. **18:** 729–746, December 1939.

The authors describe the use of kymography in the detection of myocardial infarcts, in the diagnosis of constrictive pericarditis, in pericardial effusion, and diseases of the pulmonary artery. The characteristic picture in aortic regurgitation is mentioned. The authors find the method of value in studying and teaching human cardiac physiology and in corroborating impressions of heart movement obtained by fluoroscopy. They term it a useful adjunct to fluoroscopic examination. WM. H. GILLENTINE, M.D.

The Roentgenkymogram in Myocardial Infarction: Part 1. Abnormalities in Left Ventricular Contraction. Marcy L. Sussman, Simon Dack and Arthur M. Master. Am. Heart J. **19:** 453–463, April 1940.

Part 2. Clinical and Electrocardiographic Correlation. Simon Dack, Marcy L. Sussman, and Arthur M. Master. Ibid, pp. 464–474.

Two hundred cases of myocardial infarction were examined roentgenkymographically. The following abnormalities in left ventricular pulsation were noted: (1) localized diminution or absence of pulsation; (2) complete systolic expansion or paradoxical pulsation; (3) partial systolic expansion indicated by expansion early in systole or delay in the completion of systole; (4) marked diastolic "splintering." Seventy-five per cent of the 200 cases showed these changes. They usually appear within three weeks of the attack, but may be delayed for months.

Study of the 200 cases showed that regression or disappearance of kymographic abnormalities is a good prognostic sign. All cases diagnosed electrocardiographically as myocardial infarction do not have abnormal roentgenkymograms. A small group of patients with infarcts and normal electrocardiograms may show roentgenkymographic evidence of myocardial infarction. WM. H. GILLENTINE, M.D.

Roentgenologic Demonstration of Left Ventricular Hypertrophy. George Levene and Robert M. Lowman. Am. Heart J. **19:** 401–403, April 1940.

One hundred unselected subjects were measured roentgenologically according to the technic of Vaquez for determining the thickness of the left ventricle and the measurements were compared with those reported by the pathologist at necropsy. There was an average discrepancy of 1 mm. The electrocardiograms in this series showed no correlation between left ventricular hypertrophy and left axis deviation.

WM. H. GILLENTINE, M.D.

THE DIGESTIVE TRACT

Studies of the Radiologic Image of the Gastro-Intestinal Mucosa: (A) Normal Stomach. A. Casati. Radiol. Med. **27:** 425–439, June 1940.

Casati describes a simple method by which he is able to produce excellent roentgenograms of the mucosa of the stomach. It consists in administering from 100 to 150 c.c. of an aqueous solution of colloidal barium, and making rapid roentgenograms of the stomach while the patient is prone on the table. The only difficulty in the technic is to secure a uniform distribution of the suspension over the whole stomach.

A. MAYORAL, M.D.

Roentgenologic Anatomy of Duodenal Ulcer. Guiseppe Boriani. Radiol. med. **27:** 551–556, July 1940.

Using resected specimens, Boriani made extensive studies of duodenal ulcers. A liquid suspension of barium was used over the mucosa to bring out the radiological image. The following conclusions were reached.

(1) The bulbar niche is nearly always centrally located, either in the anterior or posterior wall.

(2) The mucosal folds always converge on the niche. This, however, is difficult to demonstrate roentgenographically.

(3) The bulb frequently presents incisuras or diverticula similar to Akerlund's classical scheme, though not as often as would be expected.

A. MAYORAL, M.D.

Gastrojejunal Ulcer Following Gastro-Enterostomy Performed Twenty-Four Years Before for Pyloric Stenosis of Infancy. L. Haynes Fowler and William A. Hanson. Minnesota Med. **23:** 602, August 1940.

A young man of twenty-four had recurring attacks of epigastric pain accompanied by hematemesis and melena. He stated that he had had a gastro-enterostomy performed when he was six weeks of age. He grew to young manhood and had no symptoms relative to the stomach until six years before admission, when he was eighteen.

X-ray examination of the stomach showed a large, well functioning gastro-enterostomy with an extensive area of ulceration on the jejunal edge of the stoma. Some of the barium could be forced through the pylorus. The findings were confirmed at operation, and a reparative procedure was carried out.

The authors were unable to find an account of a similar case in the literature. PERCY J. DELANO, M.D.

Small Carcinomatous Gastric Lesions Simulating Chronic Benign Ulcer: Present Status of Differential Diagnosis and Treatment. George B. Eusterman. Minnesota Med. **23:** 703–709, October 1940.

Seventy-five per cent of gastric carcinomas are found to be inoperable. This should lead to increased efforts toward early diagnosis, and a more respectful consideration of the precursors of gastric malignancy, gastric polyps, ulcers, and chronic inflammations.

In ulcerating carcinoma the cavity produced by the ulceration is usually situated well within the normal confines of the gastric lumen. If the meniscus sign, described by Carman and Kirklin, which consists of a meniscal form of crater encircled by a transradiant zone representing the approximated overhanging border, can be demonstrated, the lesion usually proves malignant. The crater of a carcinomatous ulcer, like that of a benign ulcer, is sculptured as a niche in the gastric wall and invariably projects beyond the normal outline of the lumen. If this niche is large, above 2.5 cm. in diameter, if there is an absence of gastrospasm and of tenderness on pressure, and if the adjacent rugae are obliterated or if the margin of the niche is sharply conical or has an irregular profile, then the roentgenologist usually regards the ulcer as carcinomatous. A small carcinomatous ulcer with a small niche may have all the roentgen characteristics of benign ulcer.

Lesions near the pylorus or well up on the posterior gastric wall have a tendency to be malignant. The average diameter of carcinomas excised from the stomach at the Mayo Clinic has been 6.1 cm. Any lesion larger than 2.5 cm. is considered malignant until proved otherwise.

Patients who have had medical treatment should be periodically re-examined.

The article concludes with an evaluation of gastroscopy, age and sex incidence of benign and malignant lesions, and an operative check on the accuracy of the roentgen diagnoses. PERCY J. DELANO, M.D.

Surgical Treatment of Gastric Cancer Masquerading as Benign Disease. Waltman Walters and William H. Cleveland. Minnesota Med. **23:** 709–711, October 1940.

Regarding the malignant transformation of benign gastric ulcer, the figures of Stewart are generally accepted, namely that about 9.5 per cent of chronic gastric ulcers become carcinomatous and that 17 per cent of carcinomas of the stomach originate in gastric ulcer.

The operative procedure of choice is partial gastrectomy with removal of the lesion. The Polya type of end-to-side anastomosis posterior to the colon is utilized most frequently. The Billroth I type of anastomosis is occasionally used.

Finsterer (Proc. Roy. Soc. Med. **32:** 183–196, January 1939), in a study of 532 cases of resection for gastric ulcer, found 141 lesions to be malignant. Among the 141 were 41 which could not be distinguished grossly from benign ulcers.

Excision of the ulcer with knife or cautery without an anastomosis is occasionally done, but is not recommended, since it offers no change in the gastric physiology which would prevent a recurrence.

Partial gastrectomy has been performed at the Mayo Clinic for benign lesions with a mortality of 2.3 per cent. Benign lesions of the pars cardia may be removed with a mortality of under 4 per cent. Partial gastrectomy in carcinomatous cases was done with a mortality of 13.4 per cent.

Often the ulcerating gastric lesion appears in the roentgenogram to be located higher than it actually is, because perforation of the ulcer to the capsule of the pancreas or gastrohepatic omentum may foreshorten the stomach proximal to the lesion. In such cases the high ligation of the gastric artery, division of the gastrohepatic omentum above the lesion, and mobilization of the perforating lesion usually demonstrate an adequate amount of stomach available for resection.

PERCY J. DELANO, M.D.

Hemangioma of the Gastrointestinal Tract. Perry N. Pierose. J.A.M.A. **115:** 209–211, July 20, 1940.

Benign tumors of the gastro-intestinal tract are unusual, and vascular tumors especially rare. Hemangioma constitutes only 7 per cent of all benign tumors. Brown (Surg., Gynec. & Obst. **39:** 191, 1924) classified gastro-intestinal hemangioma as follows:

(1) Multiple tumors of vascular arcades appearing as reddish nodules situated in the submucosa and connected with an artery or a vein: (*a*) nevi, (*b*) cavernous hemangioma.

(2) Submucous tumor which grows toward the lumen of the bowel and may ulcerate the overlying mucosa by pressure and trauma.

(3) Submucosal hemangioma, which may grow to such a size that it may either obstruct the lumen of the bowel or cause a change in normal peristalsis, producing intussusception.

(4) Diffuse, ring-form hemangioma, which begins in the submucosa, involving the muscularis, constricts the lumen of the intestine, and is capable of producing intestinal obstruction.

A case is reported in which a patient suffered from telangiectatic cavernous hemangioma involving the jejunum and upper ileum. Surgical removal of the hemangiomatous portion of the intestine greatly improved her condition.

When a patient experiences repeated attacks of melena which cannot be explained by a thorough study of the history, physical manifestations, and laboratory examinations, the possibility of hemangioma of the gastro-intestinal tract should always be considered.

C. G. SUTHERLAND, M.D.

Endometriosis of the Ileum with Chronic Partial Intestinal Obstruction. Paul M. Glenn and John J. Thornton. J.A.M.A. **115:** 520–522, Aug. 17, 1940.

Endometrial implants in the ileum appear to be of unusual occurrence. In rare instances an implant in the small intestine is an incidental finding at operation, or it may prove at operation to be the causative lesion producing intestinal symptoms.

A case is reported in a white woman, aged thirty-one, who gave a one-year history of an occasional sense of fullness and vague abdominal distress after meals. The attacks increased in severity and duration. About eight months before admission the patient first noticed that they were usually associated with the menstrual period. She was admitted to the hospital with an attack that had lasted six weeks. Roentgenograms of the abdomen showed numerous distended intestinal loops with a "ladder" pattern. An enema of barium sulphate failed to reveal a colonic lesion. The Miller-Abbot intestinal tube was used to decompress the intestine, and prompt symptomatic relief followed. The advance of the tube through the lower part of the ileum was quite slow, but the tip finally stopped at the terminal ileum. Injection of a thin suspension of barium sulphate through the tube showed a narrowed area about 12 cm. from the cecum. Surgical intervention, after twelve days with the intestinal tube in place, revealed thickening and dilatation of the ileum 15 cm. proximal to the ileocecal valve. Resection of this portion with an end-to-side ileocecostomy was done. The postoperative course was uneventful. The diagnosis in a second case was made preoperatively.

C. G. SUTHERLAND, M.D.

Radiological Image of the Gallbladder after the Ingestion of Natural Medical Waters. J. M. Cabello Campos and E. B. Ribeiro. Ann. paulista. de med. e. cir. **40:** 317–323, October 1940.

The authors investigated roentgenologically the action on gallbladder function of certain so-called therapeutic waters, procured in the open market. The subjects used had been previously examined and each had shown a normal functioning gallbladder that responded to the fatty meal. The dye was administered and when a well filled viscus was seen a glass of the water was given. Follow-up films were made after 10 to 110 minutes. In no case did the writers notice any increase or decrease in the size of the organ.

A. MAYORAL, M.D.

Double Gallbladder. Fahri Isik. Klinik Radioloji. **1:** 41–43, September 1940.

Isik reports the case of a double gallbladder in a twenty-year-old woman—the two gallbladders partly overlapping in the anterior-posterior view—diagnosed by cholecystography. Duodenal ulceration was a complicating feature.

According to the author, this is the 20th case of double gallbladder reported in the medical literature.

ERNST A. SCHMIDT, M.D.

THE SPLEEN

Encysted Hematoma of the Spleen Studied Roentgenologically with the Aid of Thorium Dioxide. Consuelo Luccioni. Radiol. med. **27:** 466–471, June 1940.

Luccioni reports a case of hematoma of the spleen in which the correct diagnosis was made with the aid of thorium dioxide, 75 c.c. being injected over a period of five days. On the sixth day excellent roentgenograms of the liver and spleen were obtained, making diagnosis possible.

A. MAYORAL, M.D.

THE SKELETAL SYSTEM

Diagnosis of Intervertebral Disk Protrusion by Intraspinal Injection of Air. W. Edward Chamberlain and Barton R. Young. J.A.M.A. **113:** 2022–2024, Dec. 2, 1939.

The authors report their experience, based on more than 300 spinograms. They consider air myelography accurate and reliable, as in each case in which operation was performed the level of the lesion as previously determined was verified. Since the procedure is harmless, negative results need cause no feeling of apprehension as to unpleasant or dangerous sequelae, such as might develop if the contrast medium were an unabsorbable substance. The technic is discussed in detail.

C. G. SUTHERLAND, M.D.

Roentgenologic Diagnosis of Intraspinal Protrusion of Intervertebral Discs. John D. Camp. Minnesota Med. **23:** 688–692, October 1940.

Within the past few years a considerable literature concerning intraspinal protrusion of the intervertebral discs has appeared and the condition has been firmly established as a definite clinical and pathologic entity. Studies by Deucher and Love (Arch. Path. **27:** 201–211, 1939) indicate that the protruded fragments are composed of fibrocartilage, portions of the nucleus pulposus, and occasionally remnants of the notochord.

Visualization of the spinal subarachnoid space by use of iodized oil has become an established procedure. The chief objection to its use is that it may form a meningeal irritant. Cases must be selected, and when this is judiciously done, it is the consensus of opinion that the advantages of the method far outweigh its disadvantages.

Lumbar injection is preferred to cisternal. It is important that the iodized oil be clear, transparent, and only faintly yellow before use. Small amounts (0.2 to 2.0 c.c.) may suffice for the location of lesions that completely obstruct the canal, but it is desirable to employ larger amounts for routine use, enough to fill the subarachnoid space at any level. In Camp's experience, about 5 c.c. is the optimal amount for this purpose. With less than this, many small lesions may be overlooked.

Roentgen examination should be carried out as soon as possible after the injection of the oil. Tilting of the fluoroscopic table localizes the deformity, if it exists, in the pattern of the iodized oil. The deformity is influenced by the following factors: (1) the position of the protrusion and (2) its size, (3) associated hypertrophy of the ligamentum flavum, (4) changes in the nerve roots, such as displacement, edema, and nonfilling of affected nerve sleeve, (5) anatomic variations of the cul-de-sac.

Position of Protrusion.—Except in unusual cases the protruded fragment is situated in the anterior portion of the spinal canal and will often produce its maximum deformity when the patient is prone or prone-oblique. The classic defect is a sharply defined unilateral rounded indentation opposite an intervertebral disc.

Size of Protrusion.—Partial obstruction occurs in about 11 per cent of cases; complete obstruction in about 2.5 per cent.

Hypertrophy of the Ligamentum Flavum.—This often occurs at the same level as the protruded disc but may be found elsewhere. It produces compression of the cord posteriorly and laterally.

Changes in the Shadow of Nerve Roots.—A vertical elongated shadow of diminished density may indicate an edematous nerve root.

Anatomic Variations of the Cul-de-sac.—An anomaly may result in narrowing of the cul-de-sac below the level of the fourth lumbar intervertebral space; in other cases the cul-de-sac may terminate one or two interspaces above the usual termination, with or without a variation in its diameter.

Air myelography has been used for localization of disc lesions, but it has the following restrictions: it is unsatisfactory for non-obstructing lesions above the conus; the shadow is less distinct; roentgenoscopic verification of the defect is not possible; indeterminate examinations are frequent; determination of the amount of deformity is much less accurate.

The great majority of disc lesions occur in the lumbar or lumbo-sacral regions. Thus far, 77 per cent of negative air myelograms have been found incorrect at operation, for in this large number the surgeons have discovered operable lesions.

PERCY J. DELANO, M.D.

Protrusion of the Lower Lumbar Intervertebral Disks. W. J. Mixter and J. S. Barr. New England J. Med. **223:** 523–529, Oct. 3, 1940.

The development of recognition of intervertebral disc protrusion is reviewed and the discussion limited to the disc between the last two lumbar vertebrae and the sacrum.

The intervertebral disc is shaped like a closed horseshoe, with the weakest points posterolaterally, just anterior to the intervertebral nerve. Posterior to the nerve are the ligamentum flavum and inner border of the articular facet. The extruded fragment of intervertebral disc is an irregular mass of fibrocartilage of variable size and sometimes attached to the main body of the disc.

Rupture of the intervertebral disc occurs about ten times as frequently as thickening of the ligamentum flavum or arachnoiditis, and either of these latter conditions may give symptoms resembling a protruded disc.

The one characteristic finding of protruded disc is severe, intractable pain running down the back of the thigh and the outer side of the calf. Back pain may also be present. The outstanding physical sign is interference with straight leg-raising. The Achilles jerk is absent in 70 per cent of the patients, as is also splinting of the lumbar spine. The total protein of the spinal fluid is usually increased.

Lipiodol is advised as the contrast medium for injection into the spinal canal for study. Air is not dependable and thorotrast is believed to be too dangerous. Lipiodol should be injected only when the clinical findings are sufficient to warrant investigation.

The operative procedure for removal of a ruptured intervertebral disc is described and several good reproductions are included. JOHN MCANENY, M.D.

Neurologic Aspects of Herniated Nucleus Pulposus at the Fourth and Fifth Lumbar Interspaces. R. Glen Spurling and F. Keith Bradford. J.A.M.A. **113:** 2019–2022, Dec. 2, 1939.

On the basis of their experience with a series of 85 low intraspinal lesions treated surgically the authors present what they have found to be the characteristic clinical picture of herniated nucleus pulposus at the fourth and fifth interspaces. The significant features are severe persistent sciatic pain preceded by low back pain and associated with hypesthesia or anesthesia below the knee. If the ankle jerk is unchanged and paresthesia or hypesthesia is more anterior, including the great toe, the fourth lumbar disc is probably involved. If the ankle jerk is diminished or absent and the hypesthesia more posterolateral including the lateral aspect of the foot, the herniation is probably at the lumbo-sacral disc. The history and neurologic signs of herniated nucleus pulposus are not, however, peculiar

to this clinicopathologic entity. Neoplasm along the course of the sciatic nerve, rectal or pelvic disease, and disease of the osseous structures must be ruled out by regional and roentgen examinations before the clinical diagnosis of herniated nucleus pulposus can be made. Knowledge of the exact distribution of the fifth lumbar and first and second sacral nerves, motor and sensory, the authors consider essential to a proper clinical understanding. C. G. SUTHERLAND, M.D.

Injury to Intervertebral Discs During Spinal Puncture. M. Gellman. J. Bone & Joint Surg. **22:** 980–985, October 1940.

The frequency with which spinal puncture is used for diagnostic and therapeutic purposes and the occasional complaint of pain following this procedure prompted the detailed report of a case.

At the age of nine months, the patient had a convulsion with residual right-sided hemiparesis. There was a family history of syphilis and the patient had the disease. At twelve years, a spinal puncture was attempted, but it was a failure and was followed by severe back pain. Roentgenograms of the spine were negative at that time. Subsequently, additional films of the spine were made and the condition known as "Schmorl bodies" was demonstrated.

It is believed that spinal puncture can produce damage to the spine and that a Schmorl body or node represents the change in the vertebral body after injury. JOHN MCANENY, M.D.

Symptom of Narrowed Intervertebral Disc. H. Schaer. Schweiz. med. Wchnschr. **70:** 849–852, Sept. 7, 1940.

The author distinguishes between multiple disc narrowings and single disc narrowing. The former is a fairly common accompaniment of the degenerative arthroses of the spine, especially in advanced age; the latter always means a chronic osteomyelitis of the spine, the most common cause of which is tuberculosis. Since this is the earliest sign of tuberculosis, it is imperative that it should not be overlooked or minimized, although the accompanying deformity may be regular or irregular in character. LEWIS G. JACOBS, M.D.

Fracture of the Posterior Arch of the Atlas. Samuel E. Sinberg and Michael S. Burman. J.A.M.A. **114:** 1996–1998, May 18, 1940.

Fractures of the bodies of the lower cervical vertebrae or of the atlas are rarely followed by death. This is not true of fracture of the odontoid process, which is the mechanism of death by hanging. In fractures of the atlas, the mortality reported in a review covering a twenty-five-year period was 13.7 per cent. The rarity of fracture of the atlas is attested by the fact that only 93 cases were reported to 1938. These fractures are rarely a result of direct trauma. The usual mechanism is the pressure of the occiput on a rigid cervical spine with the muscles of the neck relaxed, conditions which usually obtain in automobile and railroad accidents. The usual fracture site is the posterior arch (unilateral or bilateral) near the lateral masses where the groove for the vertebral artery narrows the bony structure. Fractures of the anterior arch, the lateral masses, and the transverse processes are quite unusual. The history, the physical condition of stiffness and painful restricted movement of the neck, and the roentgenograms lead to the diagnosis.

Two cases of fracture of the posterior arch of the atlas are reported here. One was associated with a crushing fracture of a lateral mass and the second with a fracture of other bodies of the cervical spine.

C. G. SUTHERLAND, M.D.

Two Cases of Bilateral Congenital Dislocation of the Head of the Radius. H. Page Mauck and R. D. Butterworth. J.A.M.A. **114:** 2542–2543, June 29, 1940.

Congenital dislocation of the head of the radius is relatively rare. Keen stated that 51 cases had been recorded to 1907. McFarland in 1936 (Brit. J. Surg. **24:** 41, 1936) reported 11 cases. In 1892 Abbott (Lancet **1:** 800, 1892) described 7 cases in one family through four generations and suggested a hereditary tendency.

The present authors report two cases. In one no treatment was attempted. In the other excision of the head of the radius was done with excellent results. Operation other than removal of the head of the radius is not satisfactory. C. G. SUTHERLAND, M.D.

Solitary Plasma Cell Myeloma of Bone as an Initial Stage of Multiple Myeloma. Byron B. King. J.A.M.A. **115:** 36–38, July 6, 1940.

Some observers regard solitary myeloma of bone as a clinical and pathologic entity which is amenable to treatment and has a good prognosis. Most authorities, however, believe that it is only a matter of time before a solitary myeloma of bone becomes a multiple lesion with a fatal termination. In the course of the disease such organs as the spleen, liver, and kidneys may become involved.

The author reports a case in which a lesion of the femur was thought to be a hemorrhagic bone cyst or a benign tumor of the bone. Roentgenography revealed no other lesions of the skeleton. Biopsy showed a plasma-cell myeloma. Four years later, and five years after the onset of symptoms, multiple bone lesions were present, with a pathologic fracture of a rib.

C. G. SUTHERLAND, M.D.

Present Trends in the Study of Arthritis and Rheumatism. Russell L. Cecil. Minnesota Med. **23:** 533–542, August 1940.

Cecil begins by saying: "Rheumatoid arthritis and rheumatic fever constitute two of the last remaining unsolved problems in the field of infectious diseases."

An essential pathologic feature of rheumatoid arthritis is the presence of focal collections of lymphoid cells in the synovial membrane. Numerous agglomerations of small round cells form spherical masses in the region of the blood vessels. Cecil quotes Allison and Ghormley as follows: "Histologically the tissues show a definite picture, which is as clear cut as is that of tuberculosis, namely a proliferative change in the synovial membrane and marrow which is characterized by focal collections of lymphocytes. This microscopic picture will, we believe, be enough to establish the diagnosis of proliferative (rheumatoid) arthritis."

Dawson has shown that the subcutaneous nodule of rheumatoid arthritis differs little, if any, from the similar nodule encountered in rheumatic fever.

Focal infection plays a very doubtful rôle in the establishment of a typical rheumatoid arthritis.

Cecil's classification is as follows:

I. Infectional arthritis, of proved etiology.
II. Probably infectional, including
    Arthritis of rheumatic fever.
    Rheumatoid arthritis, with such forms as Still's disease, Marie-Strümpell form, and psoriatic arthritis.
III. Degenerative arthritis (osteoarthritis; hypertrophic arthritis).
IV. Arthritis associated with disturbance of metabolism, such as gout.
V. Arthritis of neuropathic origin, as from tabes, syringomyelia, and peripheral neuritis.
VI. Miscellaneous, as from serum sickness, hemophilia, and hydrarthrosis.

A detailed discussion of the various etiologic theories is included, with an extensive review of the experimental work now in progress, which is designed to clarify our conceptions of etiologic processes in the development of the various forms of the disease, but more particularly of rheumatoid and rheumatic fever types. PERCY J. DELANO, M.D.

Osteopsathyrosis (Lobstein Type). Paul Funk. Schweiz. med. Wchnschr. **70:** 473–479, June 1, 1940.

The author reports two typical cases of brittle bones, one with a definite familial history. He then discusses the disease at length. The patients are usually small, delicately built individuals with a well developed panniculus. Sexual precocity, commonly present, was absent in the cases reported. The blueness of the sclerae varies in intensity; an increase in the blueness during the menses occurred in the cases reported. It is apparently due to abnormal transparency of the sclerae with transmission of the color of the uveal pigment. Bony fragility does not always accompany blue sclerae. Fuss found only 107 of 177 patients with blue sclerae to have abnormal fragility of the bones, and Carrière, 125 of 267. The first fractures usually occur as the child learns to walk. This is in contrast to the intrauterine fractures which appear in osteogenesis imperfecta. Activities of the mildest sort, as picking flowers, may produce fractures in these patients. Moreover, the bones possess an abnormal tendency to bend, producing typical deformities in the femora and tibiae.

The roentgen picture is that of an abnormal porosis of the bones; the structure is prominent and the cortex thin, with a wide marrow cavity. The lengthening of the long bones is normal, in spite of the fractures, but the widening lags behind, producing long, thin bones. The epiphyses are normal. In the reported cases the porosity was more marked in the lower than in the upper extremities.

Deafness, which is believed commonly to accompany the disease, is found in only about a quarter of the cases. It is thought to be in the nature of an otosclerosis. Blue ear drums appear to be present in a high percentage of these patients, however.

In addition to the above symptoms, a certain laxness of the joints is present, which predisposes to dislocation. A generalized muscular atrophy accompanies the disease, but it is not known whether this is primary or secondary to the bone lesions.

The blood cytology is normal; the reported eosinophilia is probably due to other conditions (parasites ?) commonly present in children. The blood calcium, magnesium, and phosphorus are normal; the phosphatase is elevated, 100 per cent (Bodansky technic). The alkali reserve is normal.

Among the various causes suggested for this condition, only one can be seriously considered, and that is heredity, in view of the marked familial tendencies shown. The condition behaves like a mendelian dominant. The brittle bones and deafness, however, must be carried by different genes than the blue sclerae. The fact that the three do not always go together also negates the idea that this is a simple mutation. The whole question is somewhat confused by certain sporadic cases that behave as recessives. This may mean that we have here a complex mutation which may appear in either dominant or recessive form.

Osteopsathyrosis is often confused with osteogenesis imperfecta, but clinically there should be no difficulty in distinguishing the two, as the short deformed bones in the latter condition bear no resemblance to the long delicate ones in the former. Moreover, osteogenesis imperfecta appears at birth, while osteopsathyrosis manifests itself about the second year.

Therapy has been rather vague. Parathyroid extract, parathyroid transplantation, and removal of the parathyroids have been tried without clear-cut results. Hansen reports no benefit from thymic extracts. Ovarian and testicular preparations have given negative results also. Castration in either sex has also failed to improve the condition, although a marked increase of bony calcification has occurred. Strontium therapy has been suggested, but evidence is inconclusive. Massive vitamin D therapy, at times with calcium and phosphorus, has also been futile. The author is now trying continued small doses of these agents. No form of therapy has yet proved of value.

LEWIS G. JACOBS, M.D.

Diagnosis of Neuropathic Joint Disease (Charcot Joint): Analysis of 40 Cases. Ralph Soto-Hall and Keene O. Haldeman. J.A.M.A. **114:** 2076–2078, May 25, 1940.

The usually accepted theory of pathogenesis of the Charcot joint attributes it to a single injury or repeated injuries to an articulation which has lost its sensibility to pain. Tabes dorsalis is most frequently the underlying disease in the development of a neuro-arthropathy. A similar loss of sensibility in the joint may occur in syringomyelia, injuries to the spinal cord, and leprosy.

The characteristics of a Charcot joint, in the order of their usual appearance, are an enlargement of the joint resulting from an effusion, a relaxation of its ligaments, leading to instability and deformity on weight-bearing, thinning of the articular cartilages, marginal fractures, sclerosis of subchondral bone in some areas and atrophy of bone in other regions, loose bodies in the joint, and the formation of new bone (parosteal bone) outside the joint cavity. The most striking clinical observation is the absence of pain in the presence of such an advanced pathologic process. The positive clinical evidence of rigid pupils and absence of knee jerks is of much greater importance in the diagnosis of a tabetic arthropathy than are various tests on the blood and spinal fluid. A culture of the synovial fluid should be made prior to any operative procedure on a Charcot joint.

C. G. SUTHERLAND, M.D.

Legg-Perthes Disease of the Hip: Its Early Roentgenographic Manifestations and Its Cyclical Course. A. B. Gill. J. Bone & Joint Surg. **22:** 1013–1047, October 1940.

This is an excellent and thorough investigation of Legg-Perthes disease. The etiology is still unknown. The patient usually complains of pain in the knee at first and has a limp in the affected leg. There is limitation of motion in the hip, especially rotation. The most characteristic finding is a firm thickening of the hip found on palpation. The disease runs a cyclical course with a degenerative phase lasting about one and one-half years and a regenerative phase lasting about two to three years, which periods are shortened by the proper treatment, which consists in non-weight bearing.

It is found that slightly over-exposed x-ray films demonstrate the changes best. In the earliest phase there is necrosis in the metaphysis of the femoral neck, represented by areas of decalcification, of various shapes and sizes, that later coalesce to form a broad band of rarefaction across the entire metaphysis. Following the metaphyseal changes or accompanying them necrosis of the head of the femur occurs in an area directly overlying the earliest changes in the neck. In the head, the changes consist in irregular decalcification and fragmentation, with flattening and apparent disappearance. An early finding is an increase in the distance between the femoral head and the acetabulum, and by the same token, there is a lessening of the elliptical area formed by the inner portion of the head and the posterior lip of the acetabulum.

After a period of about one and one-half years, regeneration begins, first in the metaphysis with recalcification of the rarefied areas, and later in the head with calcification in the base, spreading toward the periphery. This phase lasts about two to three years, and results in almost perfect restitution if proper treatment is followed, but the period may be prolonged and the joint surfaces be deformed if treatment is not adequate.

Several series of illustrations show the cyclical changes in the hip. JOHN MCANENY, M.D.

Ollier's Dyschondroplasia: Report of Case. R. M. Carter. J. Bone & Joint Surg. **22:** 1063–1069, October 1940.

A forty-seven-year-old male had a deformity of the right leg dating from the first year of life. In later years he had made a satisfactory brace, with the aid of which he was able to work. The right leg extended only as far as the knee of the left leg. At the time of consultation the patient had begun to have severe pain in the heel of the short leg and because of the possibility of sarcomatous changes amputation was done.

A complete pathological report is given, but no evidence of sarcoma was found. The final diagnosis was Ollier's dyschondroplasia.

JOHN MCANENY, M.D.

Two Cases of Tuberculosis of the Os Pubis. Veysi Demirel. Klinik Radioloji **1:** 36–40, September 1940.

Tuberculosis of the os pubis is rare and is infrequently diagnosed before fistulation occurs. Abscess formation may be mistaken for femoral hernia. The author describes two cases, diagnosed roentgenologically, in patients aged twenty-four and twenty-six years.

ERNST A. SCHMIDT, M.D.

Syphilis in Bones in Infancy: Some Possible Errors in Roentgen Diagnosis. William A. Evans, Jr. J.A.M.A. **115:** 197–200, July 20, 1940.

Characteristic osseous lesions are found in a very high percentage of cases of fully developed syphilis. A child may show no osseous lesions or other evidence of syphilis at birth and after several weeks or months definite manifestations of congenital syphilis may develop. Four to six weeks are required after infection of the fetus for bone lesions to appear.

Bands of rarefaction or condensation are frequently seen at the ends of the diaphyses, especially in premature infants and those presenting nutritional disturbances. These changes cannot be regarded as specific. Periosteal shadows vary from the very fine white line parallel to the shafts of long bones, most commonly the femurs, to a heavy onion-like cloaking of the bone which may double the diameter of the shaft. Causes of these periosteal shadows are found not only in syphilis but also in subperiosteal fractures occurring at delivery, in rickets, in tuberculosis and other forms of osteomyelitis, and in a considerable group of cases in which the cause is obscure. In the latter group may be instances of hypertrophic pulmonary osteoarthropathy associated with chronic pulmonary or cardiac disease. Rickets is not often discovered in the last half of the first year of life and is recognizable by very characteristic changes at the ends of the diaphyses of long bones.

C. G. SUTHERLAND, M.D.

Aseptic Necrosis of Bone Occurring in Caisson Disease. W. A. Walker. J. Bone & Joint Surg. **22:** 1080–1084, October 1940.

This is a case report describing and demonstrating the bone changes found in caisson disease. The complete history and physical findings are presented.

Radiographically, the changes in the right hip consisted in an irregularity and flattening of the femoral head, with numerous areas of decreased density extending into the neck and surrounded by bone of increased density. The hip joint space was narrowed and the acetabulum showed arthritic changes corresponding to the changes in the femoral head. In the distal end of the femur there were irregular sclerotic changes in the medulla, without involvement of the cortex. The upper end of the right tibia showed changes like those found in the lower femur. Similar changes were suggested by the findings in several of the phalanges of the right hand. JOHN MCANENY, M.D.

Osteomyelitis Variolosa: Report of a Case. P. L. Fan. Chinese Medical Journal **57:** 571–573, June 1940.

Fan reports a case of osteomyelitis following smallpox and also reviews the literature. Of special interest are the conclusions of Eikenbary and LeCocq (J.A.M.A. **96:** 584–587, 1931) which he quotes:

(1) The virus of smallpox undoubtedly causes a non-suppurative osteomyelitis.

(2) The site of election of the virus apparently is in the epiphyseal lines of the long bones.

(3) The effect of the virus is to produce an aseptic necrosis which apparently chiefly affects the growing cartilage cells of epiphyseal lines and causes permanent closure.

In cases of obscure bony deformities in localities where smallpox is epidemic a careful history of variola in childhood should be obtained.

A. MAYORAL, M.D.

Ossifying Myositis. E. B. Ribeiro. Ann. paulist. de med. e cir. **40:** 199–222, September 1940.

Ribeiro writes a lengthy article on ossifying myositis based on personal observations and a review of the literature. He accepts the following as the most common classification:

(1) Ossifying myositis, progressive, generalized.

(2) Ossifying myositis, circumscribed, traumatic.

Eleven theories are put forth as possible explanations for the condition. Trauma in one form or another seems to be the most important single factor. Attention is called to the efficacy of roentgen therapy not only as a therapeutic but also as a prophylactic agent.

A. MAYORAL, M.D.

FOREIGN BODIES

Localization of Foreign Bodies. C. Scholder. Schweiz. med. Wchnschr. **70:** 728–730, July 30, 1940.

The author advocates locating a foreign body in the soft tissues by inserting a needle into the tissues in a direction perpendicular to the fluoroscopic screen and along the axis of the beam. This is done under fluoroscopic control so that it is possible to see when the needle makes contact with the foreign body. Depth localization is determined by "any" triangulation method, and all the surgeon has to do is follow the needle down, since it is not displaced by the traction or manipulation of the operation. The author claims originality for this idea, but one is inclined to wonder if he has tried it; it all sounds too easy.

LEWIS G. JACOBS, M.D.

OBSTETRICS AND GYNECOLOGY

Pelvicephalography: Analysis of 503 Selected Cases. William F. Guerriero, Rupert E. Arnell, and James Barrett Irwin. South. M.J. **33:** 840–844, August 1940.

An analysis is made of Ball's method of pelvicephal-

ography in 503 selected cases of pregnancy. The conclusion is that the method is often inaccurate and misleading in breech presentations, but valuable in vertex presentations. On the basis of their data the authors predict bony dystocia if the head volume exceeds the volume capacity of the inlet by at least 200 ml. or the volume capacity of the mid-plane by 250 ml.

The important measurements on the roentgenogram are the anteroposterior diameter at the pelvic inlet, that is, the true conjugate, from the pubic symphysis to the sacral promontory, and the transverse and posterior sagittal diameters at the mid-plane. The mid-plane extends from the lower border of the symphysis pubis, through the ischial spines to the junction of the fourth and fifth sacral vertebrae. Its transverse diameter is the distance between the ischial spines, and its posterior sagittal, the distance from the mid-point of the transverse diameter to the junction of the fourth and fifth sacral vertebrae.

A difficult and dangerous delivery can be anticipated in most cases in which the true conjugate is less than 9 cm. and in which the sum of the transverse and posterior sagittal diameters of the mid-plane is less than 13.5 cm. JOHN M. MILES, M.D.

RADIOTHERAPY

MALIGNANT CONDITIONS

Roentgen Therapy in Combination with Ultra-Short Waves and Insulin Injections, Especially in Carcinoma of the Stomach and Esophagus. J. G. Heeren. Strahlentherapie **68:** 444–459, 1940.

Investigations carried out by the author seem to indicate that the effect of radiation on tumors is more pronounced if combined with ultra-short wave therapy. He also gave his patients insulin (10–15 units) in an attempt to influence the permeability of the tumor cells and thus increase the radiation effect. The technic was as follows: 180 kv., 4 ma., 0.5 mm. Cu, 40 cm. focal skin distance, 330 r (in air) per field; cross-fire technic. Of the 27 treated patients, 7 had carcinoma of the esophagus, 15 carcinoma of the stomach, 1 a tumor of the mediastinum, 3 carcinoma of the lung, and 1 rectal carcinoma. All cases were far advanced and radiation therapy was given for palliative purposes. In spite of this very unfavorable material encouraging results were seen in over 50 per cent. The author recommends, therefore, that this combined treatment be given a trial.

ERNST A. POHLE, M.D., Ph.D.

Clinical Management of Breast Tumors. G. W. Taylor. New England J. Med. **223:** 538–539, Oct. 3, 1940.

In order to determine the proper procedure in the examination of breast tumors, a review of 127 cases subjected to operation was made. Patients were operated upon under general anesthesia and preparations were such as to permit additional surgery where necessary. A pathologist was usually present at the operation.

In 28 cases (22 per cent) the clinical diagnosis was in error. In 45 cases, frozen-section diagnosis was carried out, and in 36 additional cases immediate gross pathological examination was done to confirm the clinical diagnosis. In this connection it is interesting to note that of the 45 cases subjected to frozen section, 5 were erroneously diagnosed.

The conclusion is reached that no breast tumor can be termed non-cancerous on the basis of clinical examination alone, which means that every breast tumor should be biopsied. JOHN MCANENY, M.D.

Rotating Irradiation of Carcinoma of the Esophagus with Continuous Fluoroscopic Control. M. Nakaidzumi and T. Miyakawa. Strahlentherapie **68:** 254–262, 1940.

During the year 1938 the authors treated 36 patients with carcinoma of the esophagus with rotating irradiation. They developed a special technic which permits continuous control of the focusing of the radiation fluoroscopically in the darkened therapy room. Both skin and systemic reactions were found to be much milder with this type of therapy. The daily dose effective within the tumor was 500 r and a total dose of 3,000–12,000 r was given within ten to thirty days. In 32 of the 36 treated cases improvement was noted.

ERNST A. POHLE, M.D., Ph.D.

Radiation Therapy in Treatment of Metastatic Malignancy of the Chest. Roy G. Giles. South. M.J. **33:** 1033–1036, October 1940.

Although the results of irradiation of pulmonary malignant metastases are not generally encouraging, the author stresses moderate divided doses of x-rays therapeutically at fairly long intervals if the patient's general condition is fair. The alleviation of the pain, cough, and shortness of breath makes the treatment worth while, and in a few cases recovery will be obtained lasting from several months to several years.

JOHN M. MILES, M.D.

Radiation Therapy as an Aid in the Diagnosis and Treatment of Certain Abdominal Tumors. E. A. Merritt and Ralph M. Caulk. South. M.J. **33:** 989–991, September 1940.

Five cases are reported of intra-abdominal tumors which responded to external irradiation to such a degree that apparent permanent cure was obtained, life was prolonged, or an inoperable tumor became operable. The final conclusion is that the therapeutic test of irradiation is invaluable as a preliminary procedure for most intra-abdominal tumors, even though the patient may have received a hopeless prognosis.

JOHN M. MILES, M.D.

Our Results with Chaoul's Near Distance Irradiation in Gynecology. L. Csonth and Z. Horvath. Strahlentherapie **68:** 277–286, 1940.

In the authors' clinic 63 patients were treated by means of near distance radiation. Forty-three had carcinoma of the cervix, 3 carcinoma of the fundus, 3 carcinoma of the vulva, and the remaining 14 malignant lesions of other parts of the genital organs. The technic was as follows: 60 kv., 4 ma., 0.2 mm. Cu, 3–5 cm. focal skin distance, daily dose 400 r in air, total dose 4,000–8,000 r. If necessary the treatment was repeated after four to six weeks. The parametria were exposed to 180 kv., filtered with 0.5 mm. Cu + 0.5 mm. Al, through an anterior and a posterior field, with 500 r per area. The best results were seen in carcinoma of the cervix and satisfactory results were obtained in carcinoma of the vulva. No response was obtained in the patients with carcinoma of the fundus. A survey of all treated cases showed 14 patients free from symptoms and 16 definitely improved. The authors hope that with further improvement in technic better results can be obtained.

ERNST A. POHLE, M.D., Ph.D.

Dangers and Uses of Radium in the Treatment of Carcinoma of the Uterus. Maurice D. Sachs. West. J. Surg. **48:** 609–615, October 1940.

The author makes a plea for the limitation of radium therapy of uterine malignancy to the hands of those with knowledge and experience of this therapeutic medium. Complications reported in general practice, such as rectal or vesical fistulae and late recurrences, are usually due to improperly applied radium or to poor protective packing, even though proper applicators and dosage are employed. Stress is laid upon the importance of using different types of intra-uterine and colpostatic applicators depending on the gross clinical and anatomic features of the case under treatment. In checking up radium application by x-ray films, wide shifts are seen in position of the applicators between the time of insertion and the time of removal. In view of the inverse square law, one can easily see how the misapplication of radium leads to a decrease in the actual tumor dosage and to a relative increase in the amount of radiation affecting adjacent structures. The employment of properly selected radium applicators for the individual case, carefully applied by men trained in the field, is the prime requisite to good results in the radium therapy of uterine carcinoma.

SIMON POLLACK, M.D.

Treatment of Carcinoma of the Cervix. J. Markl. Strahlentherapie **68:** 347–350, 1940.

The author relates his experience with the treatment of carcinoma of the cervix by roentgen rays alone; in only a few instances was radium used as a supplementary therapeutic agent. The technical factors were as follows: 70–80 cm. focal skin distance; single dose of 300 r; total doses 8,000–10,000 r; one or two abdominal fields, four dorsal areas, two inguinal fields, one parametrial area, and occasionally an additional sacral field. Eight cases were treated in 1934, 16 in 1935, and 33 in 1936. At the time of the survey the absolute percentage of cure was 24.5; 17.5 per cent of the patients could not be traced.

ERNST A. POHLE, M.D., Ph.D.

Supplementary X-Ray Treatment for Carcinoma of the Cervix Uteri in Relation to the Direction of the Spread of the Disease. A. G. C. Taylor. Brit. J. Radiol. **13:** 293–313, September 1940.

Extension of carcinoma of the cervix usually takes place in the parametrium and in the nearby lymph nodes within the pelvis. The growth is usually predominantly unilateral, bilateral involvement occurring in only about 10 per cent of the cases.

Control of the primary lesion is the first consideration in treatment. This can usually be accomplished by radium. But as radium is usually inadequate to control extensions it should be supplemented by external irradiation. The author uses preliminary x-ray therapy only in septic and very advanced cases. He uses radium only in Stages I and II and in some cases of Stages III and IV when the lesion is symmetrical.

The use of radium plus x-ray to one side of the pelvis is advised, as the disease usually extends to one side only. For this unilateral irradiation seven small ports are used: anterior central, upper abdominal, medial abdominal, inguinal, lateral, sacral, and sciatic notch fields. All the beams are directed into the side of the pelvis to be irradiated. For the advanced and bilateral cases with general pelvic involvement, general pelvic irradiation is advised.

SYDNEY J. HAWLEY, M.D.

Advances in the Roentgen-Ray Treatment of Tumors of the Bladder. F. H. Colby and R. Dresser. New England J. Med. **223:** 565–568, Oct. 10, 1940.

In the treatment of tumors of the urinary bladder, a roentgen-ray therapy unit, operating at 1,000 to 1,200 kv., with 3.5 mm. lead filtration, at 70 cm. distance, producing 80 r per minute is used. The output is equal to that of 2,000 gm. of radium. One anterior and two posterior oblique pelvic portals are used, with a dose of 400 r per day.

This high-voltage radiation produces a greater depth dose, through small portals, and with less skin damage than with lower potentials. There is, however, a greater danger of deep tissue damage. Biopsies and frequent cystoscopic studies control the treatments. Biopsy has proved disappointing as an indication of radiosensitivity of tumors.

Only cases beyond surgical aid were treated and at least half of these showed regression of the tumor; one-third of the tumors disappeared, at least temporarily, some only to recur. The rapidly growing tumors, with little or no cell differentiation, showed the best response to treatment. The moderately malignant tumors, papillary and non-papillary, were about equally radiosensitive. Epithelial tumors of low-grade malignancy responded poorly to radiation. One case of sarcoma of the bladder was not affected by radiation. Bleeding and dysuria were frequently relieved.

JOHN MCANENY, M.D.

Treatment of Osteogenic Sarcoma. A. B. Ferguson. J. Bone & Joint Surg. **22:** 916–922, October 1940.

Ferguson has made a study of the first 400 cases of osteogenic sarcoma recorded in the Registry of Bone Sarcoma of the American College of Surgeons, to determine the survival following the various methods of treatment. His first report on this series was abstracted in an earlier issue of Radiology (**36:** 257, February 1941).

Early amputation, *i.e.*, within seven months of the onset of symptoms, was more frequent in the later years of case registration, but there was no increase in survivors. Survivors of early amputation decreased from 11.6 per cent among the first 165 cases (43 early amputations) to 2.5 per cent among the last 100 cases (40 early amputations).

The age period from eleven to twenty years produced the only survivors after early amputation, but even in this period the earlier the amputation the poorer the results. Only when the sarcoma was located in the lower femur or upper tibia was survival obtained after early amputation and here also results were poorer with the earlier amputations.

In 82 cases seen within two months of onset of symptoms, and treated by amputation, neither the age, site, variation in treatment, nor degree of malignancy explains the poorer results of early amputation. In no case did the patient survive amputation in the first month after onset of symptoms.

It is believed that the best chance of cure is offered by delay of amputation for six months, the interval to be spent in irradiation, local excision, and general building-up of the patient.

JOHN MCANENY, M.D.

Lymphosarcoma: Study of 196 Cases with Biopsy. Everett D. Sugarbaker and Lloyd F. Craver. J.A.M.A. **115:** 17, 112, July 6 and 13, 1940.

Lymphosarcoma is a malignant neoplastic disease of lymphoid tissue capable of arising in any lymphoid aggregate. It may run an acute or chronic course and is almost invariably very radiosensitive. An apparent cure is possible, but the disease is much more likely to terminate in death, at which time the wide extent of clinically unsuspected metastases may be astonishing. With Hodgkin's disease and lymphatic leukemia, it comprises a group of tumors of relatively low morbidity, the high mortality and often strikingly similar clinical

and pathologic pictures of which have properly earned them the designation "malignant lymphomas."

A study of 196 cases of lymphosarcoma, in each of which the diagnosis was confirmed by biopsy, is presented. The unit of lymphoid tissue, the lymph follicle, being composed of two distinct and easily identified types of cells (the reticulum or endothelioid cells of the germinal center, and the small lymphocytes arranged peripherally about these centers), may give rise to two corresponding histologic types of sarcoma: the reticulum-cell type, or reticulum-cell lymphosarcoma, and the lymphocytic type, or malignant lymphocytoma. The exact physiologic tie between these two cell types has not been definitely established. The not infrequent termination of the former in lymphatic leukemia strongly suggests the existence of a definite though as yet obscure relationship.

Hodgkin's sarcoma is undoubtedly a form of reticulum-cell sarcoma developing late in the course of the more chronic type of Hodgkin's granuloma or earlier in the course of the more acute forms of that disease and representing a truly neoplastic response of the endothelioid cells presumably to the exciting agent originally responsible for the chronic inflammatory reaction of the granuloma.

Any clear-cut statement of the biologic forces inciting lymphoid tissue to malignant growth is as impossible at present as it is for any other type of tissue. It must be assumed, on the basis of the histologic picture and the clinical behavior, that lymphosarcoma is a true neoplasm. Though many cases give a history of a long-standing inflammatory process in the upper respiratory tract, the rôle of tuberculosis is much less evident in lymphosarcoma than it is in Hodgkin's disease. It is not inconceivable that prolonged stimulation of lymph nodes may eventuate in malignant cellular activity.

There are no clinical features which without exception will permit a differentiation between tuberculous lymphadenitis, Hodgkin's disease, lymphosarcoma, aleukemic leukemia, leukemia, lympho-epithelioma, or even metastatic carcinoma in lymph nodes. Excisional biopsy interpreted by a skilled pathologist is imperative. The application of a therapeutic test dose of radiation is of little diagnostic value, as any of the first-mentioned conditions may respond as readily as does lymphosarcoma.

One hundred and eight patients in the authors' series received some form of irradiation: 96 were treated by roentgen rays alone; 26 received roentgen therapy plus radium; 23 were treated by radium alone; 22 patients, chiefly those with generalized disease, received Heublein total irradiation, usually combined with external local irradiation. The results are reported for 108 of these irradiated cases, constituting a so-called determinate group. Of this number, 15 (13.9 per cent) survived five or more years but only 8 (7.4 per cent) were apparently cured, 5 treated by x-rays alone and 3 by x-rays plus radium. Thirty patients were treated surgically either with or without irradiation. Six apparent cures (24 per cent of a determinate group of 25) were obtained by this treatment.

Analysis of the results suggests that for clinically early (single area of involvement) and easily accessible disease radical surgical removal followed by prophylactic external irradiation is at least worthy of consideration; for the more advanced stages local roentgen irradiation with or without total irradiation is superior to local irradiation combined with treatment by the radium pack or radium pack irradiation alone. Single doses of roentgen rays in the order of 400–500 r and total doses per field in the order of 2,000–3,000 r yielded the best results. C. G. SUTHERLAND, M.D.

NON-MALIGNANT CONDITIONS

Roentgen Therapy of Inflammatory Processes and Its Mechanism of Effect. H. Wintz. Strahlentherapie **68:** 3–16, 1940.

The author discusses briefly the history of the treatment of inflammatory disease by x-rays. He states that therapy is always followed by "shock" and describes the changes observed in the blood (leukocyte drop, delay of coagulation, drop of blood pressure, drop of colloid stability, hyperglycemia) and an increase in the tonus of the vagus. All these reactions lead to an enhanced resistance of the organism to infection. Some of the indications for x-ray therapy in inflammatory conditions are briefly discussed.

ERNST A. POHLE, M.D., Ph.D.

Varix Arterialis and Its Treatment by Radium. H Weyrich. Strahlentherapie **68:** 165–180, 1940.

The author discusses the pathologic anatomy of varix arterialis and relates the history of a case which came under his observation, a growth on the cheek of a sixty-seven-year-old woman. He treated it on the same principle as an angioma, using 2 radium screens containing 20 mg. radium each (filter 0.3 mm. Pt + a combination filter of Ag, Ni, and Pt; total thickness 1.5 mm.). The radium was applied for twenty-four hours, and two days later for an additional twenty-four hours. As photographs in the article show, the response was satisfactory. The author believes that the therapeutic effect is in all probability due to an endarteritis, although no histologic studies were undertaken in this case. ERNST A. POHLE, M.D., Ph.D.

Radium Therapy in Xeroderma Pigmentosum. J. Körbler. Strahlentherapie **68:** 181–184, 1940.

Two cases of xeroderma pigmentosum were treated by the author with radium surface applications; the response was satisfactory. Because of the ease of treatment and painlessness, he considers this the best method in this chronic disease. New lesions usually develop at certain intervals and therefore radium can be used with safety. ERNST A. POHLE, M.D., Ph.D.

Treatment of Iridocyclitis and of Vascular Diseases of the Retina with Roentgen Rays. R. Hessberg. Schweiz. med. Wchnschr. **70:** 954–958, Oct. 5, 1940.

The use of small divided doses has made it safe to employ roentgen rays in various ophthalmic diseases. The author uses either soft, medium, or hard rays, but does not define these qualities except to say that the medium are filtered with 0.5 mm. copper and 1.0 mm. aluminum.

In tuberculous iridocyclitis small doses are given, 12 to 15 r at first, increasing to a maximum of 50 r per treatment. Usually three to four treatments are given, but in no case are more than 300 r given in any year. Four cases were treated with fair results, including some improvement in sight.

In traumatic iridocyclitis preservation of vision is practically hopeless, and the aim is an esthetically suitable globe. Dosage is 100 r of medium rays every three to six days, later decreasing to 50 r. The total dose averages 350 r. Only 23 per cent of the irradiated eyes (44 cases) had to be enucleated, while in untreated cases enucleation was invariably necessary. The ocular tension returned to approximately normal levels in general. Sometimes secondary operations for improvement of sight are possible. Sympathetic ophthalmia in the sound eye as a result of irradiation is very unlikely, although it may occur in spite of the irradiation. This makes continued observation necessary.

In iridocyclitis with increased intra-ocular tension doses of 20–100 r at half-weekly to weekly intervals led to an improvement with some decrease in tension in all cases.

The most important vascular disturbance of the retina is thrombosis of the central vein. This often leads to a glaucoma hemorrhagicum. Treatment with small doses, up to 350–450 r in thrombosis of the main vessel and up to 100–150 r in thrombosis of a branch, is helpful if given early. In hemorrhage in the retina or vitreous body without thrombosis radiation is also useful; 15–25 r at eight- to ten-day intervals is indicated. One case of this sort is reported.

In other conditions the more usual treatment is to be preferred. The author concludes that radiation is a useful therapeutic agent in some ophthalmic conditions.

LEWIS G. JACOBS, M.D.

Treatment of Functional Disorders of the Female by Radiation of the Pituitary Gland. J. Kotz and E. Parker. South. M.J. **33:** 832–839, August 1940.

Two hundred and forty-three cases of scant, excessive, and painful menstruation, menopausal disturbances, sterility, and other functional disorders of the female are summarized and tabulated as regards improvement following treatment, which included organotherapy and irradiation of the pituitary region. Either 102 r weekly or 130 r every three weeks were given for three to four treatments, using 200 kv. Improvement resulted in 30 per cent of the patients with sterility, 56 per cent with amenorrhea, 63 per cent with polymenorrhea, and 70 per cent with dysmenorrhea.

JOHN M. MILES, M.D.

Effect and Dosage in the Treatment of Progressive Paralysis by Roentgen Rays. F. Voss. Strahlentherapie **68:** 498–508, 1940.

Since the first publication of Bering based on 15 cases of progressive paralysis treated by roentgen rays (see abstract in Radiology **36:** 389, 1941) 75 additional patients have been irradiated in his clinic. Total doses applied to the skull are 1,200 (small), 1,500 (medium), and 1,920 r (large) using a half value layer in copper of 1 mm. Six fields are arranged over the parietal region, frontal area, right and left sides of the skull, occipital region, and upper cervical area. The entire series is given within two weeks. Although it is difficult to explain the mechanism of the effect of radiation the author suggests that some substance or substances are released by the cells injured during irradiation which influence the pathologic process, similar to the action of roentgen rays in the treatment of inflammatory disease.

ERNST A. POHLE, M.D., Ph.D.

Recent Advances in the Treatment of Hepatic Disease. Albert M. Snell. Minnesota Med. **23:** 551–556, August 1940.

Snell reviews in detail the experimental work on liver function: removal of portions of the dog's liver, the administration of hepatotoxic substances such as carbon tetrachloride, and diversion of the portal blood by an Eck fistula or by the ligation of the common duct.

Approaching the problem of cirrhosis, he points to the improvement in therapeutic results. Maintenance of a high-carbohydrate diet and improvement in nutritional state are receiving more stress than the matter of ascites. The average duration of life after the appearance of ascites is fourteen months in the author's series, though of a group of 150 patients studied, 22 are alive after six years. A high-vitamin diet is considered important.

One phase of management is of particular interest to radiologists: that of hemorrhage from esophageal varices. Abdominal operations designed to alter the blood flow are considered too dangerous for general use; injection of these varices through an esophagoscope has been recently introduced, and is at present very promising. Administration of vitamin K to combat the prothrombin deficiency would appear to have its place.

Earlier practices such as limitation of diet, purging, and diuresis have often merely weakened the patient, and are giving way to the administration of "protective substances" in the presence of liver injury.

PERCY J. DELANO, M.D.

Treatment of Osteitis Deformans of Paget. C. Henschen. Schweiz. med. Wchnschr. **70:** 945, Oct. 5; 965, Oct. 12; 989, Oct. 19, 1940.

This is a rather complete review of the subject of Paget's disease which should be consulted in the original. Most of the material has no special bearing on radiology. In the discussion of treatment, however, the author mentions, among other modalities, irradiation with small doses. This may be given over the adrenals or over the involved bones. There is not sufficient evidence to evaluate the effects of the former; the latter has been reported to give good results by Lüdin and by Gaál. When sarcomatous degeneration has occurred the use of very heavy fractionated irradiation is recommended.

LEWIS G. JACOBS, M.D.

DOSAGE AND TECHNIC

Million-Volt Roentgen Therapy Apparatus in the General Hospital, Hamburg-Barmbeck. F. Haenisch, K. Lasser, A. Eisl, and W. Rump. Strahlentherapie **68:** 357–404, 1940.

The authors describe in this well illustrated article the million-volt apparatus which was installed at Haenisch's clinic in Hamburg. They used a Greinacher circuit and a cascade type of x-ray tube which is on ground on one end. With a current of 5 ma. and a total filter of 5 mm. lead, 150 r/min. are delivered at a focal skin distance of 1 meter. The penetration of radiation under these conditions corresponded to a half-value layer in copper of 10.2 mm. The most suitable filter material for very hard rays seems to be lead; for half-value layer determinations copper is preferable. The percentage depth dose at 10 cm. for the above quality of radiation, 60 cm. focal skin distance and a large area, was 49 per cent of the surface dose. X-rays emitted by the tube in the direction of the cathode rays have about twice the intensity and more penetration than radiation coming at a 90° angle to the direction of the cathode rays. It requires 11 cm. lead to protect against primary radiation and 0.9 cm. lead to protect against scattered radiation at 3 meters distance with a tube current of 5 ma. The energy required to produce one pair of ions in air is the same for these hard rays as for other roentgen rays excited at lower potentials. About 7 per cent of the cathode ray energy is transformed into x-rays at 1,000 kv.

ERNST A. POHLE, M.D., Ph.D.

Roentgen Therapy with Two X-ray Tubes at Potentials Between 570 and 2,000 kv. W. Ferrant. Strahlentherapie **68:** 107–135, 1940.

The author describes an ionization chamber which permits the measurement in r of very short x-rays and gamma rays. It was ascertained that the chamber was independent of the wavelength within the range used. The r equivalent for 1 mg. hr. of gamma rays of radium C was 9.2 r cm.²/mg. hr. (reduced to the filter thickness 0). The output of the high-voltage x-ray tube at 1 meter distance from the anticathode corresponded to 1.38×10^{-1} r/s; the effective wavelength was 8.7 X.E. This would correspond ap-

proximately to the gamma radiation emitted by 840 gm. of radium. For further details the original paper should be consulted. ERNST A. POHLE, M.D., Ph.D.

Percentage Depth Doses in Low Voltage X-ray Therapy. W. J. Meredith. Brit. J. Radiol. **13:** 320–321, September 1940.

The values of depth doses in contact therapy vary as much as 50 per cent as reported by various observers. These differences are due to the large amount of secondary radiation emitted by the applicator sides and the filter. If the dose at a depth of 1.0 cm. is taken as 100 per cent, and the depth doses expressed in comparison with this, the reports of various observers are in excellent agreement. It is acceptable to use the dose at 1.0 cm. depth as the standard, as the secondary radiations are so soft that they have no biological effect. SYDNEY J. HAWLEY, M.D.

Dose Measurements in Interstitial Radium Therapy. A. A. D. LaTouche and F. W. Spiers. Brit. J. Radiol. 13: 314–319, September 1940.

The authors have attempted to measure the dose in interstitial radium treatments by means of condenser ionization chambers. Measurements with micro-condensers inserted in the tissues have been made, but they are subject to so much error that the results are difficult to interpret. Measurements made at the surface near the implants are subject to less variation and occasionally measurements can be made in nearby cavities. If the lesion is in the tongue, for example, the surface measurements may be used as an index of the tissue dose.

"Elektron" condensers of the cylindrical type were employed. For some surface measurements a flat type of condenser was used, as this eliminates the error in trying to half submerge a cylindrical chamber. Measurements were made under anesthesia.

The sources of error in these measurements are the short exposure time, the difficulty in getting the half-submerged effect, and the operation of the inverse square law. The errors, however, are small and corrections can be made for them. The authors feel that the error caused by lack of equilibrium due to the wall thickness of the chamber is not sufficient to invalidate the method. SYDNEY J. HAWLEY, M.D.

Significance of the Filter and Cone Radiation in Near Distance X-ray Tubes. U. Henschke. Strahlentherapie **68:** 90–106, 1940.

Zimmer in 1938 (Fortschr. a. d. Geb. d. Röntgenstrahlen **58:** 107–110, 1938) drew attention to the fact that a soft secondary radiation is emitted by the cone and the filter of near distance x-ray tubes. Since no exact measurements of this radiation have been published, the author undertook an investigation of this problem. He used several of the machines which are manufactured for this type of therapy on the continent. It appeared that the surface dose, depending somewhat on the focal skin distance, is considerably increased, in certain instances as high as 70 per cent, by this soft radiation. At the same time, of course, the depth dose is decreased. The drop in depth dose is more pronounced for greater focal skin distances, an effect which is just the opposite of the one desired when increasing focal skin distance. In the interest of uniform and accurate dosage it is necessary for the radiologist to know whether an additional aluminum or celluloid screen has been attached to the tube in order to absorb the soft secondary radiation. It is also important that the method used for the determination of the output of near distance tubes be stated, and especially the wall thickness of the ionization chamber.
ERNST A. POHLE, M.D., Ph.D.

Dosage Distribution in Rotating Irradiation. R. du Mesnil de Rochemont. Strahlentherapie **68:** 221–253, 1940.

In this fifth communication on the same subject the author discusses the relation between focal skin distance and depth dose with special consideration of the influence of the divergence of the radiation. He also compares the rotating technic with the customary multiple field technic combined with compression. The results of his studies are illustrated in 19 graphs and sketches. In conclusion he expresses the opinion that rotating radiation therapy will be more widely used in the future, particularly because it is possible to increase the dose effective in the depth of the body without adding to the skin surface dose. For abstracts of the author's earlier papers, see Radiology **30:** 662, 1938; **32:** 372, 1939; **35:** 648, 1940.
ERNST A. POHLE, M.D., Ph.D.

MISCELLANEOUS

Dosimetric and Radiobiologic Experiments with Fast Neutrons. K. G. Zimmer. Strahlentherapie **68:** 74–78, 1940.

The author exposed a phantom consisting of a stearin-paraffin mixture to fast Li + D neutrons and to roentgen rays. The ionization produced in the phantom was measured by means of condenser type chambers. For a field of 150 sq. cm. and a distance of 23 cm. the ionization produced by the neutrons was approximately the same as produced by x-rays of a half value layer of 1.35 mm. copper. ERNST A. POHLE, M.D., Ph.D.

Contribution to the Problem of Biological Effects of Radiation. Determination of the Susceptibility of Organs to X-rays in Vivo by Means of a Biochemical Method. R. Abderhalden. Strahlentherapie **68:** 17–29, 1940.

The author studied the susceptibility of a number of organs to roentgen rays in rabbits. He found certain ferments in the urine which are capable of breaking down the proteins of the irradiated organs but not those of non-irradiated organs. If doses high enough to produce injury were applied to the hypophysis disturbances in the entire endocrine system could be noticed. With his biochemical method, which is described in the article, the author found that the lower limit of radiosensitivity of the liver is around 330 r, for the brain around 247.5 r, and for the hypophysis around 165 r. ERNST A. POHLE, M.D., Ph.D.

X-ray Reactions in the Ear of the Rabbit with Special Consideration of Variations in Intensity. H. J. Teschendorf. Strahlentherapie **68:** 304–342, 1940.

The ears of rabbits were exposed to doses of roentgen rays of 500, 1,000, 1,500 and 2,000 to 5,000 r. The erythema, pigmentation, epilation, reaction in the epidermis, reparatory processes, and late changes after single exposures were studied. The tolerance dose was 2,000 r if applied at 36 r/min. Histologic studies showed that after the macroscopic reaction had subsided there remained definite changes in the hair follicles, the epidermis, the distribution of the pigment, and in the sebaceous glands. When varying the intensities from 30 to 1,300 r/min. no changes could be detected either in the course of the reaction or in the late changes. When intensities of 7 and 1,300 r/min. were compared, slight deviations in the course of the reaction were noticeable. Experiments conducted

with fractional doses showed that the intensity has the least influence on the reaction; the order of magnitude of the fractional dose and of the total dose determined the degree of reaction. No difference could be detected in the skin reaction when comparing two types of radiations of half value layers in copper of 0.25 and 1.0 mm. ERNST A. POHLE, M.D., Ph.D.

Experimental Study on the Effect of Aniline Dye upon the Growth and Radiosensibility of Transplanted Tumor. S. Kisimoto. Jap. J. Obst. & Gynec. **23:** 68–87, April 1940.

Kato rabbit sarcoma and cultivated splenic tissue of immature rabbits were subjected to intravenous injection, injection into the tumor, or direct application of aniline dyes. The tissue glycolysis, respiration, growth, and radiosensitivity were then studied. The growth of the cultivated splenic tissues was accelerated by eosine, erythrosine, and isamine blue, especially by eosine. On the contrary, methylene blue and trypaflavine even in weak concentration prevented growth. These latter dyes also decreased radiosensitivity, while eosine, erythrosine, and isamine blue enhanced it. Thus a close relationship between the growth of tissue and its radiosensitivity was demonstrated. The fluorescence itself seemed to have no effect upon the radiosensitivity. Intravenous injections of methylene blue, eosine, and trypaflavine increased the radiosensitivity of rabbit sarcoma. This effect, the author believes, was biological, being secondary to metabolic disturbances in the cells due either directly or indirectly to dyestuffs. He concludes that since methylene blue and trypaflavine enhance the radiosensitivity of rabbit sarcoma and decrease the radiosensitivity of normal cultivated tissue, the action must be biological and not physiological. WILLIS A. WARD, M.D.

Experimental Study on the Desuetude of Renal Function by Irradiation of Roentgen Rays. Hisao Ito. Jap. J. Obst. & Gynec. **23:** 58–67, April 1940.

The author gives a detailed account of the results of irradiating healthy normal male rabbits. One group was irradiated without preliminary treatment; a second group received preliminary intravenous injections of such materials as a mercuric preparation, a mercuric preparation and grape sugar, and an iodine preparation and grape sugar. The irradiation was administered both in total dosage and in fractional dosage. Impairment or loss of kidney function was found to follow such experimentation. Excellent macroscopic and histologic illustrations demonstrate the effect on the renal tissue. The injection treatment increased the sensitivity of the renal tissue to irradiation. The mercuric preparation and grape sugar injection was found to be most effective. Irradiation in divided doses was more effective than irradiation in which the dosage was undivided. In the latter cases a lesion was also produced in the skin and intestine; this was not true when the dose was fractionated.

WILLIS A. WARD, M.D.

X-ray Anemia and X-ray Death of Small Animals. M. C. Terry and S. B. McFarland. J. Iowa M. Soc. **30:** 486–487, October 1940.

Previous experimental work has suggested that the anemia and death of small animals following sufficient exposure to roentgen rays might be due to the development of a hemolytic antibody. The authors investigated this possibility using full grown Flemish albino rabbits. They were unable to find any hemolysin in the blood serum of the animals treated.

LESTER W. PAUL, M.D.

A Nomogram for the Determination of Lead Protection against High-Voltage X-rays. W. Binks. Brit. J. Radiol. **13:** 322–323, September 1940.

A nomogram is presented which will report the amount of lead to give adequate protection when the voltage, current, and distance are given.

SYDNEY J. HAWLEY, M.D.

Photographic Reversal with X-rays. O. J. Baltner and J. E. Nafe. Physical Rev. **57:** 1048–1049, June 1, 1940.

The authors report a very simple experiment to prove that photographic reversal or so-called solarization occurs with x-rays as well as light.

In the same place Nafe and Jauncey report that pretreatment with hypo removes the solarization and that the effect does not occur when the x-ray exposure is done at liquid-air temperature.

R. R. NEWELL, M.D.

Photographic Reversal or So-called Solarization. W. F. Berg, A. Marriage, and G. W. W. Stevens. J. Optical Soc. of America **31:** 385–394, May 1941.

Photographic reversal or so-called solarization occurs only for the developable nuclei formed on the surface of the silver bromide grains in the emulsion, and does not occur for the interior of the grains. This explains its removal by a treatment with hypo before development. Apparently with very heavy exposures the silver nuclei formed on the surface of the grains recombine with bromine atoms, it being strongly suggested that the light-absorbing bromide ions are situated almost exclusively at the surface of the grain.

This appears to be connected with the "reciprocity failure," so-called, of the very high-intensity exposure, resulting in such a rapid release of electrons at the surface of the grain that further electrons are repelled. There is also a connection with the so-called Clayden effect, the desensitizing effect of a very short high-intensity pre-exposure.

The original article should be consulted because of the complicated interplay between the effect of light on the surface of the grain and on the interior of the grain in the emulsion.

R. R. NEWELL, M.D.